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# NATIONAL WORKSHOP ON RADIOACTIVITY IN DRINKING WATER

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C. Richard Cothern
Workshop Chairman

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The proceedings of the National Workshop on Radioactivity in Drinking Water contain several Committee Issue Papers. They are: (1) Regulatory Development of The Interim and Revised Regulations for Radioactivity in Drinking Water - Past and Present Issues and Problems; (2)Regulatory Consideration in Radiation Protection; (3) Health Effects and Risks from Radon-222 in Drinking Water; (4) The Occurrence of Radioactivity in Public Water Supplies in The United States; (5) Metabolism of Ingested Uranium and Radium; (6)Cancer Risk from the Lifetime Intake of Radium and Uranium Isotopes; and (7) Compliance and Policy Issues and Recommendations Related to Revision of the National Interim Primary Drinking Water Regulations for Radionuclides. These papers were offered to the U.S. Environmental Protection Agency in the form of recommendations.

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U.S. Environmental Protection Agency

# NATIONAL WORKSHOP ON RADIOACTIVITY IN DRINKING WATER

Workshop Chairman: C. Richard Cothern
Health Effects Branch
U.S. Environmental Protection Agency
Washington, DC

Workshop Coordinator: Sheri E. Marshall
Dynamac Corporation
Rockville, MD

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# REGULATORY DEVELOPMENT OF THE INTERIM AND REVISED REGULATIONS FOR RADIOACTIVITY IN DRINKING WATER - PAST AND PRESENT ISSUES AND PROBLEMS

William L. Lappenbusch
C. Richard Cothern

# REGULATORY DEVELOPMENT OF THE INTERIM AND REVISED REGULATIONS FOR RADIOACTIVITY IN DRINKING WATER - PAST AND PRESENT ISSUES AND PROBLEMS*

William L. Lappenbusch and C. Richard Cothern
Office of Drinking Water
U.S. Environmental Protection Agency
Washington, D.C. 20460

## Abstract

Developing the Revised Regulations for Radioactivity in Drinking Water under the Safe Drinking Water Act requires information from all areas and disciplines related to this endeavor. As one step in the regulatory process, the background and history of that process as it applies to radioactivity in drinking water is described. The issues involved in developing the Revised Regualtions are detailed in the following areas: monitoring and sources of exposure, dose evaluation, health effect, engineering, economics and general policy development. This paper thus was prepared for use at the National Workshop for Radioactivity in Drinking Water held at Easton, MD, May 24,26, 1983.

## INTRODUCTION

The 93rd Congress passed Public Law 93-523 known as the Safe Drinking Water Act (SDWA) , on December 16, 1974. This law has subsequently been amended on November 16, 1977 (Public Law 95-190), September 6, 1979 (Public Law 96-63) and December 5, 1980 (Public Law 96-502). The purpose of the Act was to amend the Public Health Service Act to assure that the public is provided with safe drinking water.

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*The views expressed in this paper are those of the authors and do not necessarily reflect the views and policies of the U.S. Environmental Protection Agency.

Section 1412 of the SDWA instructed the Environmental Protection Agency (EPA), among other things, to propose and promulgate first the interim regulations and later, the revised regulations for hazardous constituents in drinking water (organics, inorganics, microorganisms and radionuclides). The EPA was instructed by Congress to work with the National Academy of Sciences (NAS), or another equivalent body, and propose recommended maximum contaminant levels (RMCLs) for undesirable pollutants in drinking water as part of the revised regulations. The RMCLs are to be based on health and to take into consideration the impact of:

"(A) The existence of groups or individuals in the population which are more susceptible to adverse health effects than the normal healthy adult;

(B) The exposure to contaminants in other media than drinking water (including exposures in food, in the ambient air, and in the occupational settings) and the resulting body burden of contaminants;

(C) Synergistic effects resulting from exposure to or interaction by two or more contaminants;

(D) The contaminant exposure and body burden levels which alter physiological function or structure in a manner reasonably suspected of increasing the risk of illness."

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Congress also instructed EPA to promulgate revised national primary drinking water regulations which specify a Maximum Contaminant Level (MCL), or to require the use of treatment techniques for each contaminant for which an RMCL is established if monitoring is not technically and economically feasible. An MCL is a standard based on health, taking cost occurrence and practicality into account. The health effects include both carcinogenic, non-carcinogenic, fatal, and non-fatal effects; the costs include those directly due to health effects as well as those needed to cover monitoring and treatment. As an adjunct to formal MCL's for drinking water contaminants, the Office of Drinking Water (ODW) sometimes develops Health Advisories (HA). These guidance levels have no legal standing; however, they can provide useful information to assist public water systems when an unregulated contaminant is detected.

The NAS did not provide EPA with RMCLs for most of the radionuclides that may occur in drinking water as dictated by Congress (they did provide some discussion for uranium). Therefore, this National Workshop for Radioactivity in Drinking Water was particularly important in the potential standard-setting process because it provided a public forum for discussion of a large number of technical issues that will be considered by EPA in its rulemaking activity. The Office of Drinking Water assembled knowledgeable experts at this workshop to address issues relating to radioactivity in drinking water in such areas as (1) occurrence, (2) sampling and analytical methods, (3) metabolism and dosimetry of high linear energy transfer (LET) radioisotopes, (4) health effects and risks due to the ingestion of naturally occurring alpha particle emitters, (5) preliminary thoughts about radon ranging from intake(s), uptake(s) and resulting bioeffects, (6) treatment, waste management and associated costs, and (7) compliance and policy issues. The information will be evaluated very carefully by EPA as the ODW prepares the standard-setting criteria for radioactivity in drinking water.

## INTERIM REGULATIONS AND CONTROL MEASURES

### A. OVERVIEW OF INTERIM REGULATIONS (NIPDWR)

On August 14, 1975, EPA proposed the interim primary regulations for radioactivity in drinking water. The National Interim Primary Drinking Water Regulations (NIPDWR) for radionuclides were promulgated in their final form on July 9, 1976 (Federal Register, Vol. 41, No. 132, pgs. 28404-28409). In 1979, a Variance and Exemption (V&E) report was prepared for all regulated constituents in drinking water including radionuclides (USEPA79). Also in 1979, suggested guidance based primarily on health was provided for natural uranium to the State of Colorado, Congressmen, Bureau of Indian Affairs, other governmental bodies and private citizens upon their request (La79). A more formalized Health Advisory for uranium was submitted to EPA's Science Advisory Board (SAB) in 1983.

The interim regulations address natural and man-made radioactivity as shown in the flow diagrams in Figures 1, 2 and 3 and described in more detail below. For natural radioactivity, control measures were established for gross-alpha-particle-activity, Radium-226 (Ra-226) and partially for Radium-228 (Ra-228). Natural uranium and radon were specifically and intentionally excluded.

GROSS-APLHA-PARTICLE-ACTIVITY, RA-226 AND RA-228

I. Maximum Contaminant Level (MCL)

- A. Ra-226 and Ra-228 = 5 pCi/l where detection limit = 1 pCi/l
- B. Gross-alpha-particle-activity (including Ra but excluding U and Rn) = 15 pCi/l where detection limit = 3 pCi/l

II. Monitoring procedures (for all community water supplies - ground and surface)

- A. Initial sampling
    1. Sampling initiated June 24, 1979
       Analysis completed by June 24, 1980
    2. Compliance based on analyses of an annual composite of four consecutive quarterly samples or average of analyses of four consecutive quarterly samples
    3. Overall method of compliance:
        - a. Perform gross-alpha-particle-activity analysis.
        - b. If gross-alpha-particle-activity > 5 pCi/l, perform Ra-226 analysis.
        - c. If Ra-226 > 3 pCi/l, perform Ra-228 analysis.
    4. At discretion of State, data collected within one year prior to the effective date of the regulations (June 24, 1977) could have been substituted for the "initial sampling".

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B. Follow-up sampling

1. Suppliers must repeat the complete initial sampling process once every four years unless radioactivity is < 1/2 MCL's whereby a single sample once every four years is sufficient at the discretion of the State.

2. Suppliers must monitor more frequently if contamination is likely only when the State orders them to do so.

3. Suppliers must repeat the complete initial sampling process again and within one year following (a) introduction of new water source, (b) contamination, (c) major change in distribution system, or (d) major change in treatment processing.

4. Suppliers must monitor source water in addition to free-flowing tap water if two or more sources exist with significantly different levels of radioactivity.

5. Suppliers need not monitor for Ra-228 if analyzed in the initial sampling.

6. Suppliers must complete annual monitoring of any water supply where Ra-226 > 3 pCi/l or when ordered by the State.

7. When the average annual MCL for Ra or gross alpha particle activity is exceeded, supplier must notify State and public. Quarterly sampling is required until problem is resolved.

BETA AND GAMMA RADIOACTIVITY FROM MAN-MADE RADIONUCLIDES

I. MCL

A. Four mrem/year to total body or critical organ with a variety of detection limits.

II. Monitoring procedures (for all surface water supplies supplying 100,000 people or more or those designated by the State as being impacted by a nuclear facility)

A. Initial sampling

1. Completed by June 24, 1979.

2. Compliance based on analysis of a composite concentration of four consecutive quarterly samples or an average of analyses of four quarterly samples.

3. Overall method of compliance:
    a. Perform gross beta analysis.
    b. If gross-beta-particle-activity $< 50$ pCi/l, then must check for $^{3}H < 20,000$ pCi/l and $^{90}Sr < 8$ pCi/l. If both $^{3}H$ and $^{90}Sr$ are present, sum annual dose equivalent to bone marrow (see figure 2).
    c. If gross-beta-particle-activity $> 50$ pCi/l, then radiochemical analysis is required and critical organ dose must be calculated. Dose must not exceed four mrem/year.
4. Suppliers may be required by the State to conduct additional monitoring to determine the concentration of man-made radioactivity in principal watersheds.
5. Suppliers using only ground water may, at the discretion of the State, be required to monitor for man-made radioactivity.
6. Data collected within one year prior to the effective date of the regulations (June 24, 1977) may be substituted for the initial sampling.

B. Follow-up Sampling
1. Suppliers must monitor via initial sampling techniques at least once every four years.

C. Special requirements for man-made radionuclide contamination of drinking water by nuclear facilities (see figure 3).
1. Suppliers must initiate quarterly monitoring (average of analyses of three monthly samples or analysis of a three month composite sample) of gross beta particle activity.
    a. If gross beta particle activity $> 15$ pCi/l, then analyze for $^{89}Sr$ and $^{134}Cs$. If gross Beta $> 50$ pCi/l, then conduct radiochemical analysis and calculate total body or critical organ dose. Dose must not exceed four mrem/year.
2. Suppliers must collect and analyze a composite of five consecutive daily samples each quarter for $^{131}I$. State may require more frequent monitoring when $^{131}I$ is present in finished water.
3. Suppliers must monitor annually for $^{90}Sr$ and $^{3}H$ whereby four quarterly samples are analyzed either as a composite or individually and averaged.

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4. State may allow substitution of environmental surveillance data taken in conjunction with a nuclear facility for direct monitoring by the supplier, himself.

D. If average annual MCL for man-made radioactivity is exceeded, supplier must notify State and public. Sampling must then occur on a monthly basis until problem is resolved.

In response to the proposed interim regulations of August 14, 1975, five major issues surfaced and were considered in promulgating the NIPDW regulations in 1976:

(1) The number and location of the public water systems impacted by the proposed maximum contaminant levels for radionuclides.

(2) The number and location of water supplies requiring radium analysis at the proposed 2 pCi/liter gross-alpha-particle-activity screening level.

(3) The estimated preliminary assessments of the costs and technology for radium removal.

(4) The validity and appropriateness of an aggregate dose method for setting maximum contaminant levels.

(5) The acceptability of a maximum contaminant level for radium of 5 pCi/liter as opposed to a higher or lower level.

With respect to the number of public water systems impacted, EPA estimated at that time that approximately 500 of the nation's community water systems would exceed the 5 pCi/l MCL for radium. The proposed 2 pCi/l gross-alpha-particle-activity screening level to determine if analysis for Ra-226 was needed was increased to 5 pCi/l to avoid a relatively large number of water supplies from conducting a rather expensive radium analysis. EPA did recommend but not require, however, that in localities where Ra-228 may be present in significant quantities, the State establish a screening level no greater than 2 pCi/l. Treatment methods including ion exchange, reverse osmosis and lime-softening were recommended for consideration as possible methods for lowering the concentration of radium in drinking water. At that time, operating data from municipal water treatment systems indicated that average radium removal efficiency via the ion exchange cycle ranged from 93-97%. Concern was expressed that operating personnel at some treatment plants would be exposed to radiation. The EPA made a limited evaluation of exposures to operating personnel working in the vicinity of ion exchange units and determined that their exposure levels could be in the range of 25-100 mrem/yr. This was well below the Federal occupational guide for radiation workers of 5,000 mrem/yr. Concern about inadequate waste management practices also became an issue and EPA committed itself to address this problem area by the time the revised regulations were proposed.

EPA considered the question of whether or not a small community would be required to adhere to the interim regulations even if the aggregate dose for that particular community is small. It was decided that the individual risk rates are useful tools to protect public health in small communities, and population risk values are useful in determining overall national priorities in standard-setting. EPA also chose to assume a linear, non-threshold relationship between the magnitude of the radiation dose received at environmental levels of exposure and ill health. This policy was adopted in conformity with the generally accepted prudent assumption that there is some potential ill health attributable to any exposure to ionizing radiation and that the magnitude of this potential ill health is directly proportional to the magnitude of the dose received. In adopting this general policy, EPA recognized the inherent uncertainties, especially at low radiation doses. Furthermore, EPA acknowledged that at environmental levels it may well be impossible to statistically prove via epidemiological studies that radiation in drinking water causes cancer. The Agency nevertheless believed that the policy was a prudent one.

The 5 pCi/l total radium concentration was accepted as the most appropriate level to protect public health considering cost and feasibility. Using the National Bureau of Standards' Handbook 69 (NBS63), it was calculated that if one consumed two liters/day at 5 pCi Ra-226/l over a lifetime, the radiation dose to the bone would be approximately 150 mrem/yr. Furthermore, using the BEIR I report (NAS72), an excess cancer risk rate of 100 cases/$10^6$ people exposed/lifetime was estimated. It should be remembered, however, that animal studies (Do69) showed that Ra-228 has a measured biological effectiveness over twice as great as that of Ra-226, so that the calculated excess cancer risk rates for Ra-226 may well have underestimated the total risk of 5 pCi/l of Ra-226 and Ra-228 combined.

## B. CONTROL MEASURES

The variance and exemption (V&E) levels for man-made radioactivity in drinking water were prepared in 1979 and are essentially the same guidances as that finalized on April 5, 1979, following the Three Mile Island (TMI) incident. It is unlikely that variances nor exemptions will be necessary because of the remoteness of an accident. Even then only variances for tritium ($^3H$) should be granted. Following the TMI accident, two offices within EPA, namely the ODW and the Office of Radiation Programs (ORP), jointly issued "Drinking Water Alert Levels":

I. Screening Level

| | |
|---|---|
| Gross beta with iodine precipitated | 40 pCi/l |
| Gross beta without iodine precipitated<br>(separate radioiodine tests may be needed) | 100 pCi/l |

II. Alert Levels

50 mrem/year
(12 times EPA 4 mrem/year standard*)
10 mrem dose commitment for any one day

*(See National Interim Primary Drinking Water Regulations, USEPA76)

Current Federal Guidance for transient rate of intake provides limitations on food and water intake that are comparable to an annual dose equivalent of 50 mrem/year and contain a recommendation that for transient situations the dose should be averaged over one year (26 Federal Register 9057, 1961). IT IS HEREIN RECOMMENDED THAT THIS ANNUAL DOSE RATE, 50 MREM/YEAR TO ANY ORGAN, BE USED AS AN ALERT LEVEL FOR RADIOACTIVITY IN FINISHED DRINKING WATER.

For continuous intake, concentrations of a single man-made radionuclide yielding 50 mrem/year to any specific organ can be found by multiplying by 12 the concentration listed in reference USEPA76 or USEPA81. When more than one radionuclide is present, instructions for limiting their sums are given in reference USEPA76.

For continuous intake, an annual dose rate of 50 mrem results from each of the following concentrations.

| | |
|---|---|
| Strontium-90 | 96 pCi/l |
| Strontium-89 | 960 pCi/l |
| Cesium-137 | 2400 pCi/l |
| Iodine-131 | 36 pCi/l |

For drinking waters having concentrations of radionuclides above the alert level, a determination on use should depend on radiochemical identification. IT IS FURTHER RECOMMENDED THAT THE INDIVIDUAL DOSE COMMITMENT FROM ONE DAY'S INTAKE (2 LITER) SHOULD NOT EXCEED 10 MILLIREM. A 10 mrem dose commitment results from the following concentrations:*

| | |
|---|---|
| Strontium-90 | 7,200 pCi/l |
| Strontium-89 | 72,000 pCi/l |
| Cesium-137 | 180,000 pCi/l |
| Iodine-131 | 2,700 pCi/l |

Proper use of the alert level should keep annual doses below 50 mrem/year and is applicable to a clean-up operation lasting as long as one year.

In the case of natural radioactivity, EPA used the guidance provided the President in 1961 which was specified by the Federal Radiation Council (FRC) for transient intakes (FRC61). For Ra-226 and Ra-228 the V&E level is 10 pCi/l. This level was chosen since the upper limit of the FRC Range

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*Concentrations causing a 10 millirem dose commitment are 900 times those given in Appendix B of NIPDWR (USEPA76).

II guide was 20 pCi/day via food and water. Above this range, evaluation and application of additional control measures is always necessary (26 FR 9057, 1961). Provided that a comparable intake of radium via the food pathway is unlikely, exemptions for water supplies containing less than 10 pCi/l or a dose equivalent of 300 mrem/yr would be compatible with FRC Guides. Occassionally, exemptions for concentrations exceeding 10 pCi/l for strictly limited times, may be acceptable. Exemption, but not variance, is provided for gross alpha up to 30 pCi/l using the same justification as provided for radium.

In granting V&E schedules for compliance, the following areas are considered: the source of exposure, extent to which the MCL is exceeded, type of radionuclide and amount present, number of people exposed, duration of exposure, both past and future if no remedial action should occur, other sources of exposure, and other sources of water. When treatment methods are available, compliance schedules should encourage early installation of treatment processes or encourage the water supplier to find and use an alternate source of water.

Guidance concerning the health effects of uranium in drinking water was developed after receipt of requests from the State of Coloradok in 1979, and several other governmental bodies for health advice on uranium in drinking water. A suggested guidance of 10 pCi/l or 20 pCi/day of natural uranium was proposed (La79).

The suggested guidance for uranium was calculated using the present interim standard for radium as a comparable level. The radium level was used for comparative purposes because (1) no acceptable uranium chemical toxicity data were available, (2) a lifetime number as opposed to a 1-day, 10-day or longer-term transient exposure would be needed since uranium is a natural, not a man-made constituent and lifetime exposures are commonplace, (3) the radiotoxicity of radium is rather well known, and (4) both radium and uranium are bone seekers with similar distribution patterns in bone.

In order to calculate the suggested guidance for uranium, the concentration of natural uranium, consumed over a lifetime in two liters of drinking water/day which would result in approximately the same dose and risk to the bone and bone marrow as radium was determined. Using ICRP # 2 (ICRP 59) and a composite risk estimation (relative and absolute) established by the Office of Radiation Programs, USEPA, it was estimated that 5 pCi Ra-226/l results in a 150 mrem/yr bone dose which could cause an estimated 0.7-3 bone and other cancers per million people exposed per year which is equivalent to an excess cancer risk rate of about 100 cases per million people exposed ($10^{-4}$ risk rate) per lifetime. It was determined that exposure to 10 pCi natural uranium/l resulted essentially in the same dose and risk of cancer; thus, the suggested guidance of 10 pCi/l.

Since that time, the Office of Drinking Water has reviewed its approach and estimates of risk using a modified ICRP 30 model (Su81). Assuming uptake values of $f_1 = 0.2$ and $f_2' = 0.2$ as suggested by ICRP 30 (ICRP 79), a quality factor of 20 for high-LET alpha radiation and assuming that the activity of U-238 and U-234 are equal, an endosteal bone and red bone marrow dose equivalent of 100 mrem/yr was calculated for the lifetime consumption of 10 pCi $U_{nat}$/l at an ingestion rate of 2 l/day (Co83a). Using EPA's linear hypothesis, this dose rate might cause 34 excess cases/million people exposed/lifetime (Table 1). These calculations of dose equivalents and risk rates for uranium remain essentially equivalent to those recently calculated for radium, namely 92 mrem at the 70th year and a cancer risk rate of 44 excess cases/lifetime/million people exposed to 5 pCi Ra-226/l at 2 l/day (Table 2). Thus, the comparison of 5 pCi Ra/l and 10 pCi U/l in terms of dose equivalency and cancer rates remain essentially the same.

The National Academy of Sciences (NAS83) has recommended a chronic Suggested-No-Adverse-Response-Level (SNARL) for uranium based only on chemical toxicity. They assumed a minimum-observed-effect-level of 1 mg/kg/day, an uncertainty factor of 100 and that a 70 kg adult consumes 2 l/day of water which provides 10% of the daily uranium intake. They calculate the chronic SNARL as follows:

$$\frac{1\ \text{mg/kg} \times 70\ \text{kg} \times 0.1}{100 \times 2\ \text{liters}} = 35\ \text{micrograms/liter}$$

At equilibrium they observe that this would be equivalent to 11.6 pCi U/l. The EPA does not necessarily endorse this calculation.

## REVISED REGULATIONS

Under the SDWA, one of the significant steps in the regulatory process is the promulgation of the Revised National Primary Drinking Water Regulations (NPDWR). These revised regulations will contain several parts including an MCL based on possible adverse health effects and feasible treatment technology while taking cost into consideration. These regulations shall be amended whenever changes in technology, treatment techniques and other means permit greater protection of the health of persons, but in any event such regulations will be reviewed at least once every three years.

It should not be surprising if the revised drinking water regulations for radioactivity differ from the interim regulations. This "open-minded" approach at this juncture would seem warranted, not because the interim regulations are good or bad, too strong or too weak, but because;

(1) our understanding of the occurrence of Ra-226, gross-alpha-particle-activity, and uranium in drinking water has been substantially enhanced,

(2) our concern for radon has increased because of its presence and its quite significant population risk, and

(3) the nuclear industry's ability, responsibility and effort to properly build, operate, decommission/store nuclear facilities, devices and/or their by-products is becoming more evident.

The ODW, EPA, via this workshop activity identified problem areas that need resolution prior to the completion of our criteria for development of the revised regulations. What follows is a list of some of the issues and areas of concern to EPA. The issues discussed here are divided into the five areas of exposure, dose, health effects, engineering and economics and policy.

## A. MONITORING AND SOURCES OF EXPOSURE

In order to develop regulations for radioactivity in drinking water, ODW must determine the sources of exposure and body burdens from natural and man-made radionuclides in drinking water, food and air as well as the concurrent exposure in the workplace. Exposure to other contaminants that would be expected to result in similar or other mechanisms of stress should also be identified, quantified or at least estimated.

Sampling and analytical methods are needed to adequately monitor the occurrence of radionuclides in drinking water. This area may seem straight-forward and complete but that is not necessarily the case. Several issues need exploration including (1) use and abuse of "screen" monitoring, (2) frequency of monitoring for select radionuclides, (3) need for inclusion of additional analytical techniques, (4) identification of the appropriate detection limits, (5) impact of high dissolved solids on alpha counting, (6) use or misuse of the fluorometric method for analyzing uranium, and (7) the design of our Laboratory Certification program.

Naturally occurring radionuclides like U-238, U-234, Ra-226, Rn-222, Pb-210 and Po-210 and daughter products are part of The Uranium Series. Th-232, Ra-228, Th-228, Ra-224, Rn-220 and daughters are members of The Thorium Series and also contribute to the body burden. Another set of naturally occurring radionuclides is called The Actinium Series, but this Series appears to be less significant in our discussions here. Many of these important radionuclides exist in drinking water primarily via ground as opposed to surface water sources.

The abundance of isotopes in natural uranium is such that U-238, U-235 and U-234 are present 99.27, 0.72 and 0.006%, respectively. The mass ratio of U-234 to U-238 is small but at equilibrium their activities are equal. The hexavalent state is particularly important in water because almost all tetravalent compounds are practically insoluble.

Using a recent ORNL Report (Dr81) and the paper by Cothern and Lappenbusch (Co83b) entitled "Occurrence of Uranium in Drinking Water in the U.S.", EPA estimates that:

(1) Average natural uranium concentration in surface, ground and domestic water is 1, 3 and 2 pCi/l, respectively. Domestic sources are those that could be used for drinking water purposes, but are not necessarily now in use.

(2) The highest population weighted averages are geographically lying between the States of Montana and Texas, and California and Kansas. South Dakota, Nevada and New Mexico have average values of 6.7, 4.3 and 2.9, respectively. The

States of California, Wyoming, Texas, Arizona and Oklahoma all seem to have values between 2.5 and 2.7.

(3) The population weighted average for the U.S. is 0.8 pCi/l.

(4) The U-234/U-238 ratio for most water supplies seems to be between 1 and 3, with surface waters in the U.S. being closer to equilibrium than that of ground water. The ratio can range up to 10-15.

(5) Of the 60,000 some community water supplies, 25-650 would exceed a uranium concentration of 20 pCi/l, 100-2,000 would exceed 10 pCi/l and 2,500-5,000 would exceed 5 pCi/l. This estimate is based on those supplies designated as domestic supplies.

(6) The average U.S. citizen exposed to uranium ingests 1,460 pCi/year (85%) via drinking water and 240 pCi/year (15%) via food. The concentration of uranium in the atmosphere is responsible for only a minor part of one's intake.

Using the compliance monitoring data specified in the interim regulations as a reference point, it appears that some 500 community drinking water supplies exceed the 5 pCi Ra/l. This estimate is based primarily on Ra-226 data rather than Ra-228 data since the interim regulations did not require monitoring for Ra-228 unless Ra-226 levels exceeded 3 pCi/l. About 170 public water supplies are known to have Ra-226 concentrations greater than 5 pCi/l and about 350 more are expected from those that have not reported yet (Co83c). Problem areas include Illinois, Iowa, North Carolina, South Carolina, Georgia and a few other states. It should also be pointed out that many homes use private wells which are not subject to NIPDWR for their water supply and a significant number contain Ra-226. Thus, the human exposure to radium in drinking water will be larger than that indicated here.

Human exposure to radon comes from the earth's crust and is in our ambient outdoor environment. It may become concentrated in homes. One vehicle for transport into the house is via the water distribution system. The drinking water concentrations in the U.S. range from a few pCi/l to a few 100,000 pCi/l in the Northeast, with the average somewhere between 500-5,000 pCi/l. In areas where radon is high, private wells probably deliver more radon into the household than do community water supplies.

Locations of the larger deposits of thorium in the United States has been published by Staatz, et al. in 1979 (St79) and the U.S. Geological Survey appears to be studying its presence in the U.S. Significant deposits have been found in Montana, Idaho, Colorado, Wyoming, California, and Alaska in the West; Illinois and Wisconsin in the Midwest; and North Carolina, South Carolina, Florida and Georgia in the East. However, thorium is highly insoluble in water and its occurrence is thus limited.

Our occurrence data base for Ra-228, the U-234/U-238 ratio, Rn-220 and Rn-222 and their daughters Th, Pb-210 and Po-210 is insufficient. There seems to be two methods ODW can use to fill in the data gaps: (1) more monitoring or

(2) predictive modelling. Perhaps strategic monitoring to support and verify predictive models would enable us to successfully estimate the amount of one or more members of the Uranium Series or the Thorium Series when our understanding of one or more other members of each series is known via existing data. For example, if we know how much U-238 is present, could we predict how much Ra-226 and/or Rn-222 should be there as well? This type of predictive modelling will require a substantial understanding of geochemistry, aquifers, etc. Furthermore, could we predict qualitatively and quantitatively Ra-228 in water supplies? The reward for accurate predictive modelling could be high and could eliminate substantial monitoring costs for those water suppliers not predicted to have a particular radionuclide(s).

ODW needs to determine the relative source contribution from food, air and water for radium, uranium and radon. The indoor exposure of radon via the water distribution system needs delineation. Occupational exposures must also be taken into consideration. Good occurrence data or estimates will be required to predict population risk where population risk equals the product of the population concentration times the risk/concentration or better yet the population dose times the risk/dose. Population risk estimations are useful in setting priorities for standard-setting but are not necessarily useful in determining whether a small community should undertake remedial action because of a contaminated water supply.

## B. DOSE EVALUATION

An examination of the pharmacokinetics and dosimetry of both natural and man-made radionuclides is required because of (1) the introduction of ICRP Report No. 30 (ICRP79) in place of ICRP Report # 2 (ICRP59), (2) both uranium and radon (and their daughters) are under consideration for regulation, (3) concentrations associated with a specific dose rate (i.e., 4 mrem/yr) must be established for the man-made beta and photon emitters, (4) variance and exemption levels must be proposed for radionuclides in drinking water, and (5) protective action levels (PAGs) will ultimately be required, if not sooner, should an uncontrollable situation arise unexpectedly.

Several points need clarification and delineation:

(1) $f_1$ (gut to blood) and $f_2'$ (blood to bone) for Ra-226 vs. Ra-228 (and daughters)

(2) $f_1$ (gut to blood) and $f_2'$ (blood to kidney and bone) for U-238 vs. U-234

(3) differential deposition of uranium and radium in the bone

(4) impact of nutrition on uranium and radium deposition

(5) differential retention and biological half-life of uranium and radium in critical organs (bone and bone marrow)

(6) differential dose rate of uranium and radium to the bone and bone marrow

(7) different pharmacokinetic patterns and dose estimation following experimental vs. environmental exposures

(8) appropriateness of extrapolating animal pharmacokinetics data to humans

(9) intake, uptake, distribution, retention and metabolism of radon and its daughters via ingestion, inhalation and their combination

(10) impact of other sources (house air, environmental air, occupational setting) upon the total committed dose via drinking water

(11) relationship between exposure and organ dose and dose rate as complicated by age

(12) impact of tobacco smoking on the lung dose via radon daughters

## C. HEALTH EFFECTS

In order to properly identify the RMCL, the adjusted Acceptable Daily Intake (ADI) for non-carcinogen bioeffects and non-threshold carcinogenic effects, the health effects data base must be reexamined. The extrapolation process must be reviewed and the major issues associated with making these scientific judgments must be discussed. Points for consideration/discussion include:

(1) Identification and validation of the linear dose response curves for natural radioisotopes (i.e., Ra-226, Ra-228, U-238, U-234, radon and its daughters), man-made beta and photon emitters

(2) Comparative toxicities (Ra-226 vs. Ra-228, U-238 vs. U-234, radium vs. uranium)

(3) Use or abuse of the threshold and "practical" threshold concepts

(4) Suitability of the BEIR III (NAS80) report for use in determining the health effects of radionuclides in water

(5) Chemical toxicity of uranium vs. its radiotoxicity

(6) Calculation of an adjusted ADI for natural uranium using non-carcinogenic data

(7) Validity of safety factors

(8) Use of NAS' toxicological analysis of uranium

(9) Recognition and calculation of the impact of multiple stressors

At this point, ODW favors the following positions on those issues stated above; however, ODW is open to constructive suggestions towards possible modification:

(1) For standard-setting purposes, the linear, non-threshold dose/response curve is most prudent

(2) Ra-228 is 2-3 times more radiotoxic than Ra-226. U-238 and U-234 are roughly equivalent in toxicity. Uranium is one-half as toxic as radium

(3) The threshold concept is not prudent for calculating excess cancer risk rates, especially for genotoxic, human carcinogens. The "practical" threshold concept has not been proven or shown to be a viable concept

(4) BEIR III is a useful guide for appreciating the health risks of radioactivity

(5) Chemical and radiotoxicity of uranium may be of the same order of magnitude numerically but the organ systems affected are much different

(6) An Adjusted ADI cannot be calculated for uranium, based on chemical toxicity data until better dose/response data become available

(7) Use of NAS' safety factors for non-carcinogenic data would be appropriate; however, consideration must also be given to bioaccumulation, experimental design and multiple stress interaction

(8) NAS' estimate of the toxicity of natural uranium should be reconsidered

(9) When more than one carcinogen is present, additivity must be assumed especially if the same organ system is impacted directly or indirectly

In regards to multiple stress toxicology and environmental standards/health advisories, two basic issues need attention (Fig 4).

(1) Dose/response studies need to be designed multifactorially and must account for chemical, biological and physical stressors; all acting concurrently prior to, during and after the experimental exposure or insult

(2) Health effects guidance and the control of multiple stressors (inorganics, organics or radionuclides) in drinking water should appreciate the concepts of additivity, synergism and antagonism

Co-insult studies should be preceeded by a detailed appreciation of "baseline stress", where "baseline stress" means the normal quantifiable and transitory changes that occur in an animal who has been subjected only to necessary environmental stimuli. "Baseline stress" takes into account the normal quantifiable changes in blood hormone levels, WBC and RBC density in the peripheral blood, or mitotic indices, weight of an organ, or any quantifiable parameter which may change as a result of the time of day, sex or age, etc. An understanding of fluctuation in measurable parameters in the experimental animal is imperative to obtaining good data (La77). Also significant is gaining an understanding of corresponding variability of similar parameters for humans.

To illustrate the point (Fig. 5), male Chinese hamsters were subjected to various x-ray doses at five different times of the day or night (La72). In this study, $LD_{50/30}$ values for animals exposed at 9:00 AM, 1:00 PM, and 3:00 PM were essentially identical while $LD_{50/30}$ for 11:00 AM and 1:00 AM were found to be significantly different: that is, 823 rads and 954 rads, respectively. This paper also reported that there is a true linear correlation between $LD_{50/30}$ values and mitotic indexes and that radiosensitivity for the Chinese hamster is dependent upon the time of the day. This study clearly pointed out the importance of circadian rhythm on radiosensitivity. The effect of circadian rhythm on radiosensitivity is not unique to terrestrial organisms. Some aquatic animals including the rough-skinned newt (Taricha granulosa) are circadium rhythm dependent as far as radiosensitivity is concerned (La70).

## D. ENGINEERING AND ECONOMICS

Lime softening, ion exchange and reverse osmosis have been demonstrated as methods for removing radium from drinking water (USEPA77). Although EPA is currently conducting laboratory and field studies of methods for removing uranium from drinking water, these studies are incomplete. Methods being investigated for uranium removal include anion exchange, lime softening, and reverse osmosis. It thus remains to be seen if a method for uranium treatment is both available and can be operated at a reasonable cost. Two methods appear possible for the removal of radon from drinking water; viz, aeration and adsorption by granular activated carbon. More information is needed on the feasibility and cost of these methods. Possible methods for removing man-made radionuclides from drinking water are mixed bed ion exchange resins and reverse osmosis. The feasibility and costs involved in these and other possible methods need to be determined.

Removal of radioactivity from drinking water produces waste in the form of brine from ion exchange, lime softening sludge and the reject stream from reverse osmosis. Some issues that relate to these and other potential wastes are:

(1) Is disposal of such wastes already prohibited by some States?

(2) How is the waste to be classified - high level, low level, source material, other?

(3) How is this problem related to the Resource Conservation and Recovery Act (RCRA) and Superfund regulations and activities?

(4) What criteria should be used to protect the public?

(5) What criteria should be used to protect workers at the treatment plant?

(6) How should the radioactive waste be disposed of?

(7) What costs are incurred in waste management practices involved in the treatment of drinking water for radioactivity?

## E. GENERAL DEVELOPMENT

The ODW would encourage input in the area of interpretive issues for the control of radioactivity in drinking water. Issues of particular importance include:

(1) Should Health Advisories be developed and if so, under what conditions?

(2) How can predictive modelling which would estimate that radionuclides would not be expected in geographic portions of the U.S. be used in implementation and design of regulations?

(3) What factor should be used to determine appropriate monitoring frequencies?

(4) Since uranium is both chemical and radiotoxic, how does one quantifiably determine an RMCL?

(5) Should ODW assume linearity between dose and effect?

(6) Does the concept of de minimus risk have application in the design of drinking water regulations as developed under the Safe Drinking Water Act?

(7) Should the health basis of standards be based on individual risk, population risk, or both?

(8) Should natural radionuclides be regulated individually and/or via the umbrella concept as man-made radionuclides presently are controlled in the NIPDWR of 1976?

(9) If a composite RMCL approach is acceptable, how should the question of synergism be addressed?

(10) Since Congress instructed EPA to take other sources of exposure into account when setting drinking water RMCL standards, should EPA set aside a specific exposure allotment for drinking water after weighing, of course the dose from air, food and the occupational setting?

(11) Should exposure of treatment plant operators be a factor in the setting of standards for radioactivity in drinking water?

(12) How should the drinking water standards consider waste management problems?

(13) How should public notices be presented in view of the public's perception of radioactivity?

(14) What are the training needs at national, state and local levels?

(15) What elements and input should be considered in analyzing the risks of radioactivity in drinking water and determining what an acceptable risk is? (see the following discussion).

The estimation of risk for drinking water contaminants requires the knowledge of the occurrence, the population exposed and the individual risk. Figure 6 shows how this information inputs to the risk estimation process.

In general, there are limits to knowledge about any subject and the current state of knowledge about the health effects of contaminants in drinking water has its limits. Since it is not possible to estimate risk with 100% accuracy, it is often expressed as a range of values.

Some of the complexities involved in determining an acceptable risk rate level are whether the risk involved is: voluntary or involuntary, ordinary or catastrophic, natural or man-made, immediate or delayed, continuous or occasional, controllable or uncontrollable, old or new, clear or unclear, necessary or a luxury, temporary or permanent, fatal or debilitating, curable or uncurable, equitable or unequitable, reversible or irreversible, long or short biologic half life. The way in which the public perceives all of these variables contributes to the complexity of choosing an acceptable risk rate level. Although we are generally a risk aversive nation, there is a wide variety of attitudes, responses and value judgments to these complexities.

Several Federal agencies and offices have determined risk rate levels to be used for standard setting purposes. The EPA's Office of Water Regulations and Standards considers risks in the range of $10^{-7}$ to $10^{-5}$ cases per lifetime as a target. The U.S. Supreme Court in its recent decision involving benzene (July 2, 1980) stated that "if the odds are one in a thousand ... a reasonable person might well consider the risk significant and take appropriate steps to decrease or eliminate it." In the same decision, they also stated that "If, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant." These observations have been taken from dicta and thus are out of context, but they do give an idea of the possible bounds. The Food and Drug Administration considers that $10^{-6}$ cases/lifetime a "virtually safe" level.

Only a few federal standards allow lifetime risk levels as high as $10^{-4}$ cases per lifetime, according to conservative high dose/low dose extrapolation models. The MCL for trihalomethanes in drinking water and the vinyl chloride air standard are among those that do. When the radium MCL was promulgated, it was thought to be of that same order. After careful examination of recent model calculations, however, the excess risk of about $0.4 \times 10^{-4}$ cases/lifetime may be more accurate.

In view of the uncertainties in scientific data and the variety of value judgments involved in evaluating risks, it appears that there is no single, universal and systematic method for determining acceptable risk. From the above discussion, standards have been set by government that result in individual risks in the range of $10^{-4}$ to $10^{-6}$ cases/lifetime. For humans the most acceptable risk rate level is zero. But we do not live in a risk free society and thus it is possible to estimate a number that could be considered in the standard setting process to protect humans from unnecessary risk.

The above discussion concerns the individual risk rate level. Although it is important to develop such a value for the individual it is important to know the risk that the entire population is exposed to, commonly known as population risk. The population risk can be calculated by multiplying the population dose/concentration by the risk per dose/concentration.

## DISCUSSION AND CONCLUSION

From the list of issues in the areas involved in the development of the Revised Regulations for Radioactivity in Drinking Water, it should be clear that they are many, varied and interrelated. It should be realized that the issues described here are those that relate to the scientific, technological areas with some discussion of the cost involved. In the process of regulatory development, inputs from several other areas will be needed. Some of these additional areas include: more economic input including cost/benefit analysis, psychological analysis of perception, fear and other emotionally related phenomena, political aspects on the Federal, State and local levels, social and moral aspects and others. Thus the information provided by this national workshop on Radioactivity in Drinking Water is only part of that needed to develop and promulgate the Revised Regulations for Radioactivity in Drinking Water.

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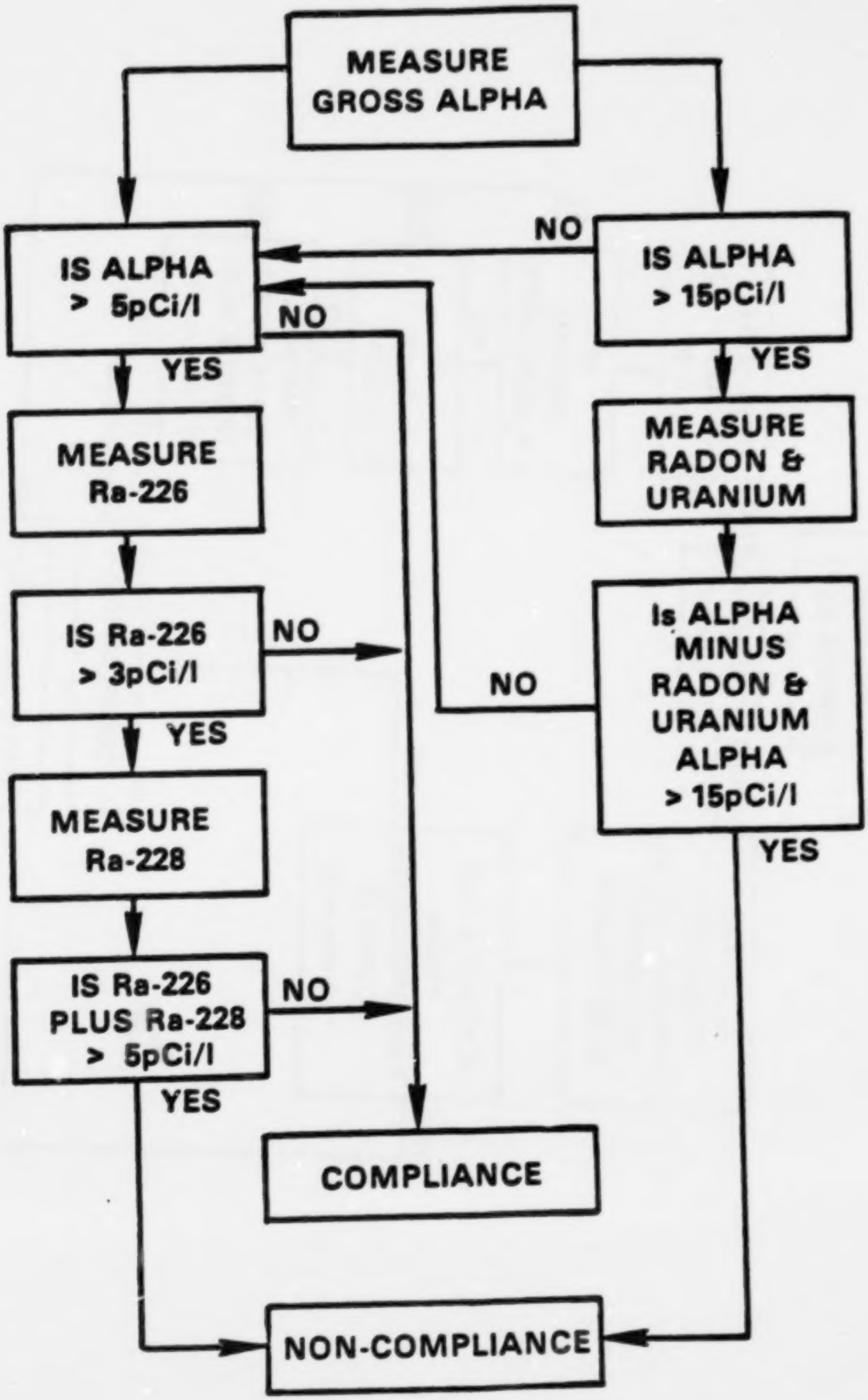
## Figure Captions

| Figure | |
|---|---|
| 1 | Flow chart for gross-alpha-particle-activity monitoring (U.S. EPA, Las Vegas, Environmental Monitoring and Support Laboratory). Note that the interim regulations do not require that radon and uranium be measured if the gross alpha activity is greater than 15 pCi/l. |
| 2 | Flow chart for gross-beta-particle-activity monitoring for a water source not designated as being contaminated by effluents from nuclear facilities serving more than 100,000 persons as designated by the State. (U.S. EPA, Las Vegas, Environmental Monitoring and Support Laboratory) |
| 3 | Flow chart for monitoring drinking water samples near a nuclear facility. (U.S. EPA, Las Vegas, Environmental Monitoring and Support Laboratory) |
| 4 | Interrelationship between factors in a multiple stress situation. |
| 5 | Survival of 80-day-old, young adult, male Chinese hamsters, weighing 20-26 g each, after exposure to various x-ray doses at five different times of the day or night. Percentage survival was calculated using data through Day 30. Sample size ranged from 10 to 30 animals point. (reproduced from La72) |
| 6 | Flow chart showing the input of information for the risk estimation process. |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IS ALPHA > 15pCi/l
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YES
YES
MEASURE Ra-226
MEASURE RADON & URANIUM
IS Ra-226 > 3pCi/l
NO
NO
Is ALPHA MINUS RADON & URANIUM ALPHA > 15pCi/l
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MEASURE Ra-228
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IS Ra-226 PLUS Ra-228 > 5pCi/l
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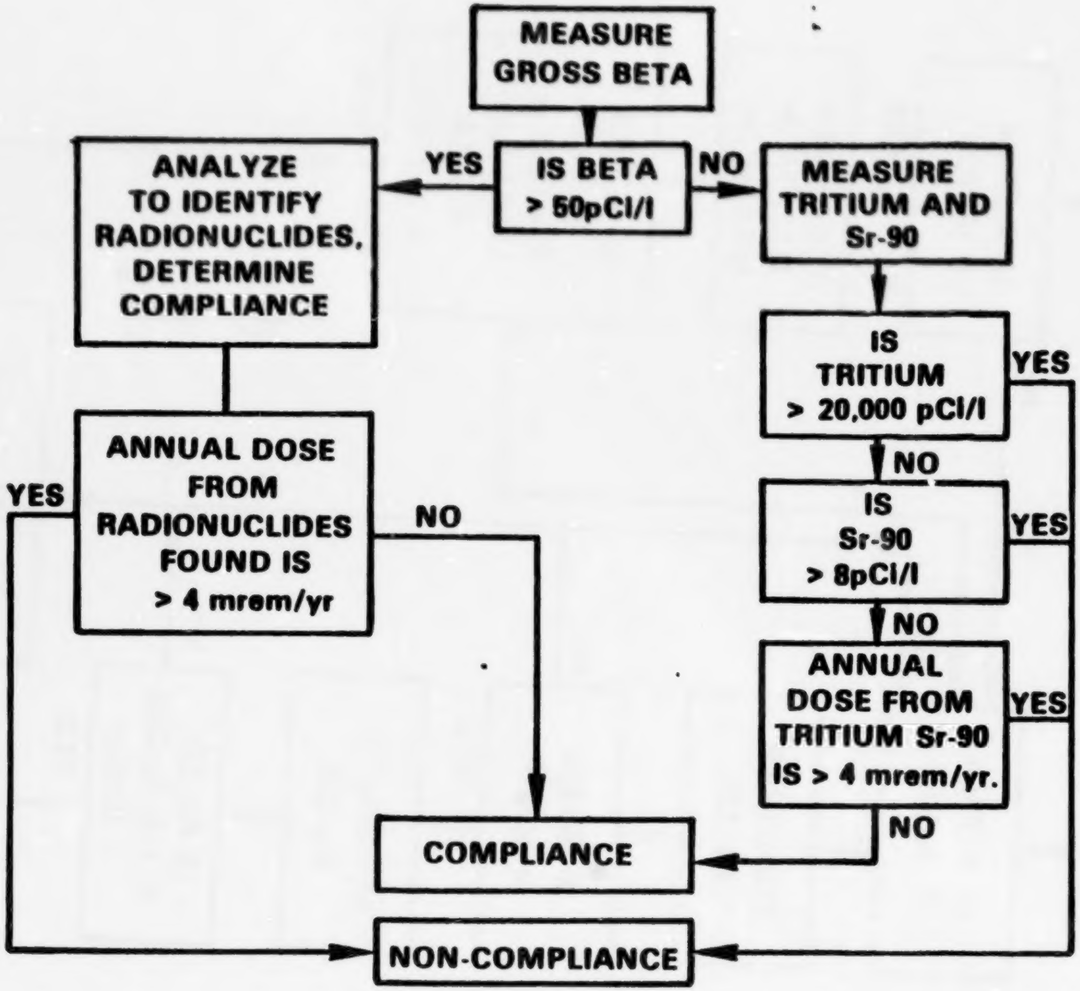
YES
COMPLIANCE
NON-COMPLIANCE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IS BETA > 50pCI/I
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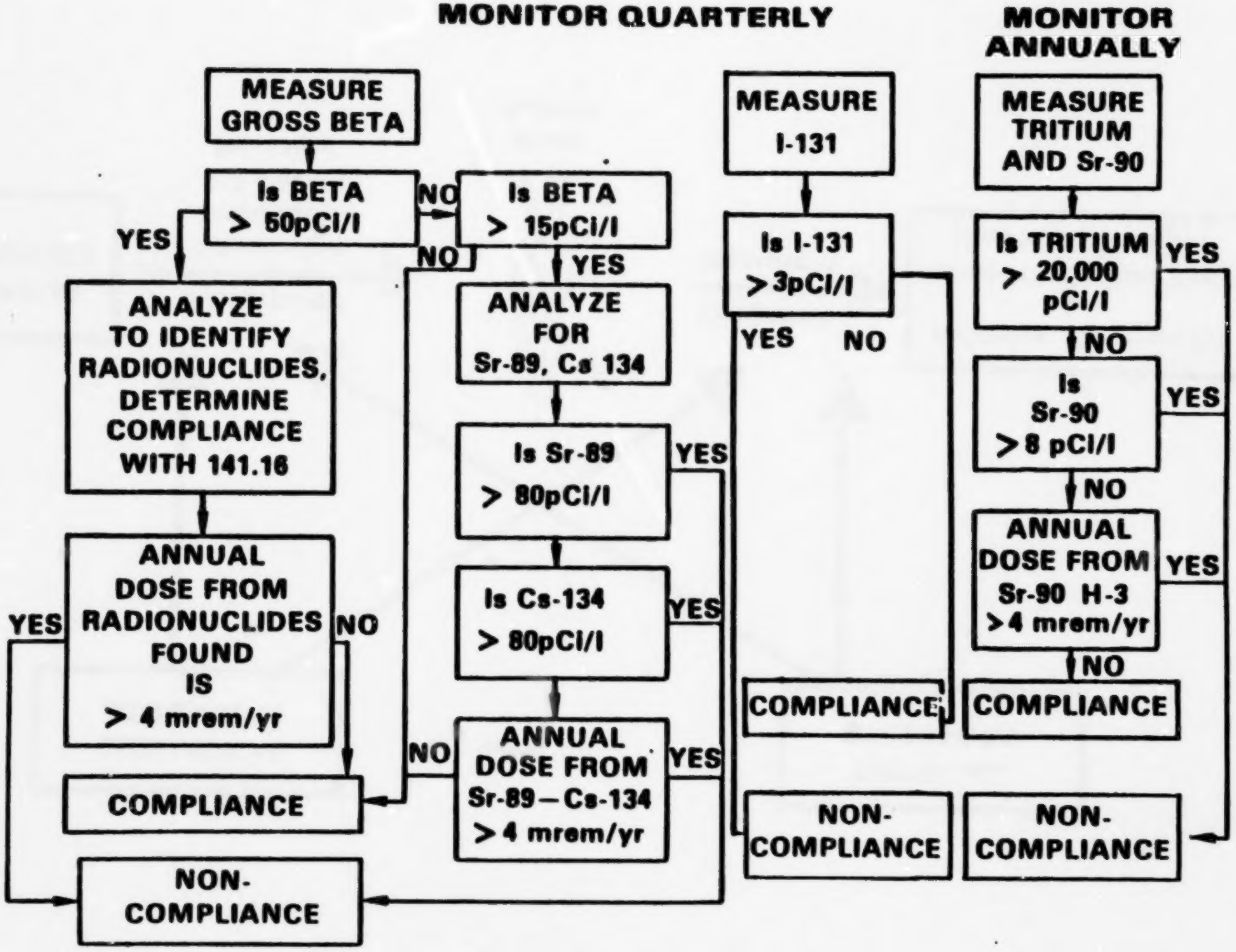
IS TRITIUM > 20,000 pCI/I
YES
NO
IS Sr-90 > 8pCI/I
YES
NO
ANNUAL DOSE FROM TRITIUM Sr-90 IS > 4 mrem/yr.
YES
NO
ANNUAL DOSE FROM RADIONUCLIDES FOUND IS > 4 mrem/yr
YES
NO
COMPLIANCE
NON-COMPLIANCE

Figure 2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MONITOR ANNUALLY
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ANNUAL DOSE FROM RADIONUCLIDES FOUND IS > 4 mrem/yr
YES
NO
COMPLIANCE
NON-COMPLIANCE
Is BETA > 15pCI/I
NO
YES
ANALYZE FOR Sr-89, Cs 134
Is Sr-89 > 80pCI/I
YES
Is Cs-134 > 80pCI/I
YES
ANNUAL DOSE FROM Sr-89 — Cs-134 > 4 mrem/yr
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YES
MEASURE I-131
Is I-131 > 3pCI/I
YES
NO
COMPLIANCE
NON-COMPLIANCE
MEASURE TRITIUM AND Sr-90
Is TRITIUM > 20,000 pCI/I
YES
NO
Is Sr-90 > 8 pCI/I
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ANNUAL DOSE FROM Sr-90 H-3 >4 mrem/yr
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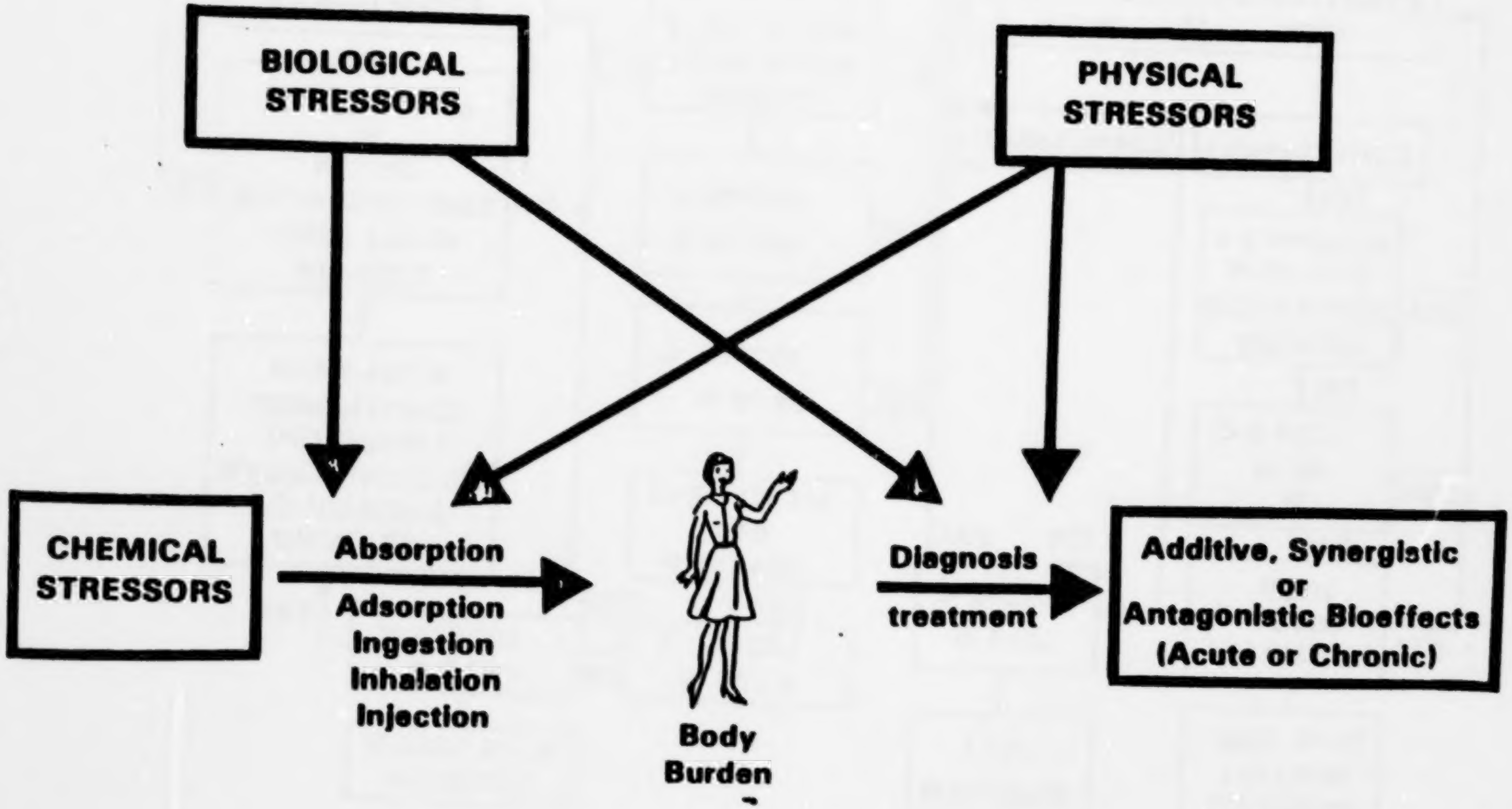
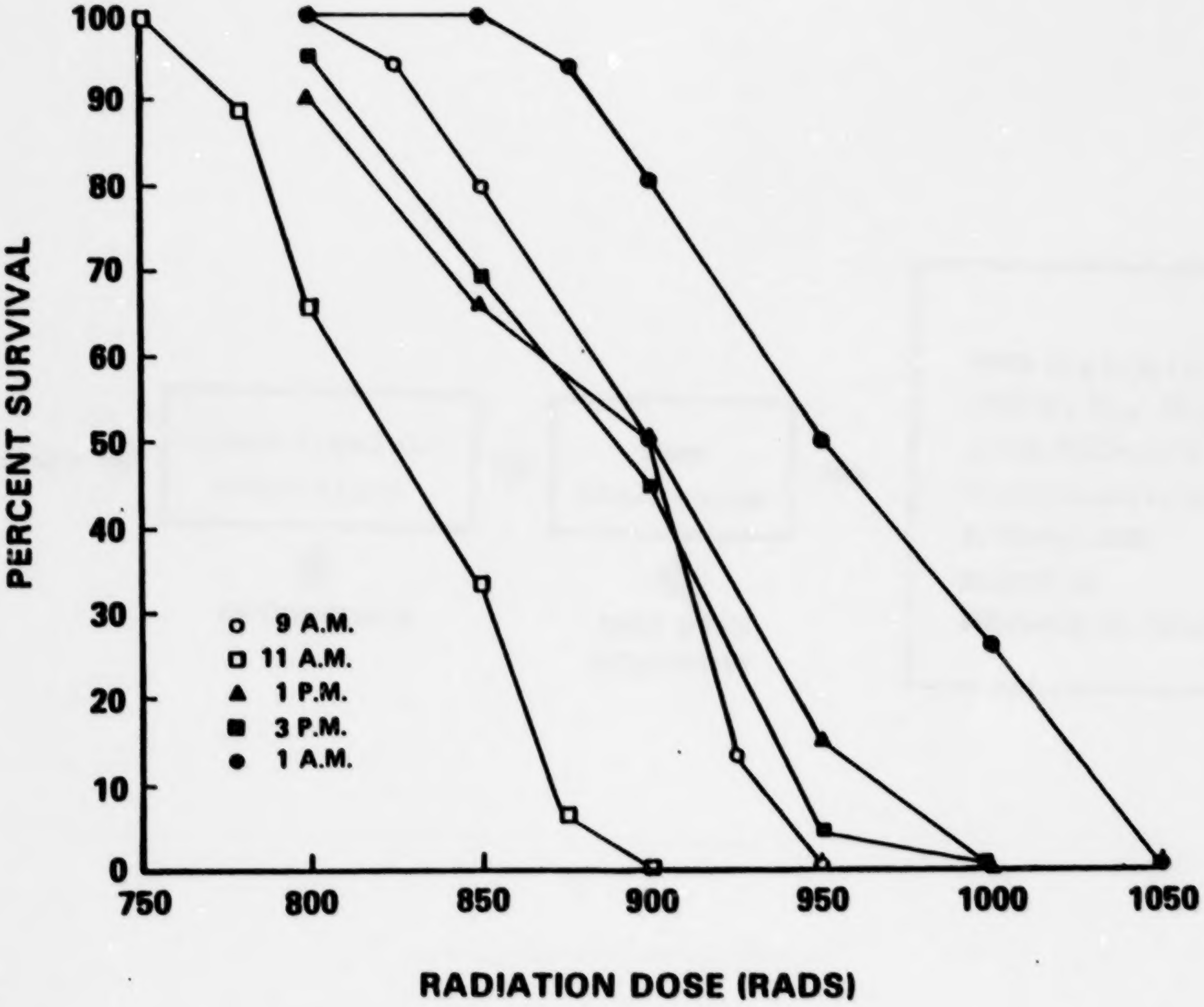
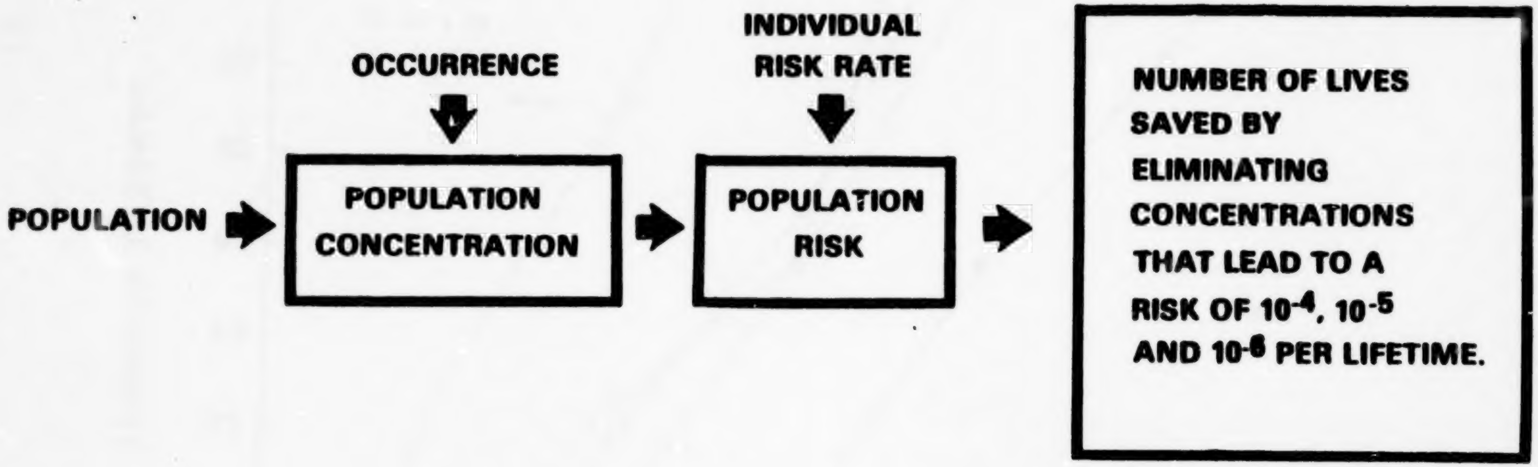
NO
COMPLIANCE
NON-COMPLIANCE


BIOLOGICAL STRESSORS
PHYSICAL STRESSORS
CHEMICAL STRESSORS
Absorption
Adsorption
Ingestion
Inhalation
Injection
Body Burden
Diagnosis
treatment
Additive, Synergistic or Antagonistic Bioeffects (Acute or Chronic)


PERCENT SURVIVAL
100
90
80
70
60
50
40
30
20
10
0
750
800
850
900
950
1000
1050
RADIATION DOSE (RADS)
○ 9 A.M.
□ 11 A.M.
▲ 1 P.M.
■ 3 P.M.
● 1 A.M.



POPULATION

OCCURRENCE

POPULATION CONCENTRATION

INDIVIDUAL RISK RATE

POPULATION RISK

NUMBER OF LIVES SAVED BY ELIMINATING CONCENTRATIONS THAT LEAD TO A RISK OF $10^{-4}$, $10^{-5}$ AND $10^{-6}$ PER LIFETIME.

## Table Captions

| Table | |
|---|---|
| 1 | Dose Equivalent and Risk Rate Estimate for the Ingestion of Uranium |
| 2 | Dose Equivalent and Risk Rate Estimate for the Ingestion of Radium |

Table 1

| Isotope | Organ | Risk[a] | Dose Equivalent Rate[b] |
|---|---|---|---|
| $^{238}U$ | endosteal bone | 8.9 | 84 |
| | bone marrow | 12.0 | 5 |
| | all others | 10.3 | - |
| $^{234}U$ | endosteal bone | 11.1 | 103 |
| | bone marrow | 16.6 | 7 |
| | all others | 9.3 | - |

a = Number of premature deaths per lifetime per million people exposed for ingestion of 20 pCi/day (50% via each isotope)

b = Dose equivalent rate in mrem/yr at 70 years from ingestion of 20 pCi/day

Source: USEPA, Office of Drinking Water. Health Advisory for Uranium.

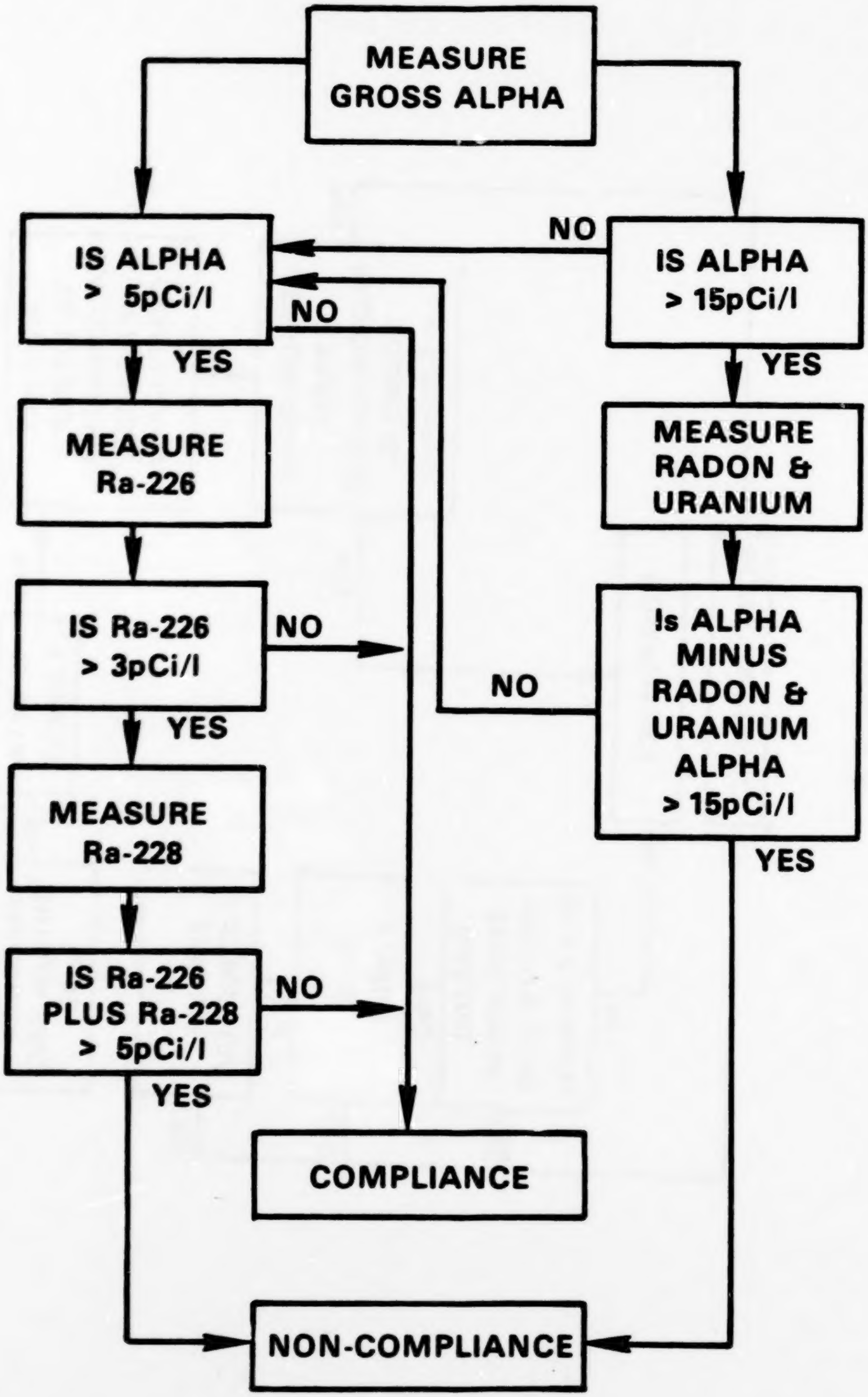
Table 2

| Isotope | Organ | Risk[a] | Dose Equivalent Rate[b] |
|---|---|---|---|
| $^{226}Ra$ | endosteal bone | 8.5 | 83 |
| | bone marrow | 30.8 | 8.6 |
| | all others | 5.1 | - |

a = Number of premature deaths per lifetime per million people exposed for ingestion of 10 pCi/day

b = Dose equivalent rate in mrem/yr at 70 years from ingestion of 10 pCi/day

Source: USEPA, Office of Drinking Water. Health Advisory for Uranium


MEASURE GROSS ALPHA
IS ALPHA > 5pCi/l
IS ALPHA > 15pCi/l
NO
YES
MEASURE Ra-226
MEASURE RADON & URANIUM
IS Ra-226 > 3pCi/l
Is ALPHA MINUS RADON & URANIUM ALPHA > 15pCi/l
MEASURE Ra-228
IS Ra-226 PLUS Ra-228 > 5pCi/l
COMPLIANCE
NON-COMPLIANCE

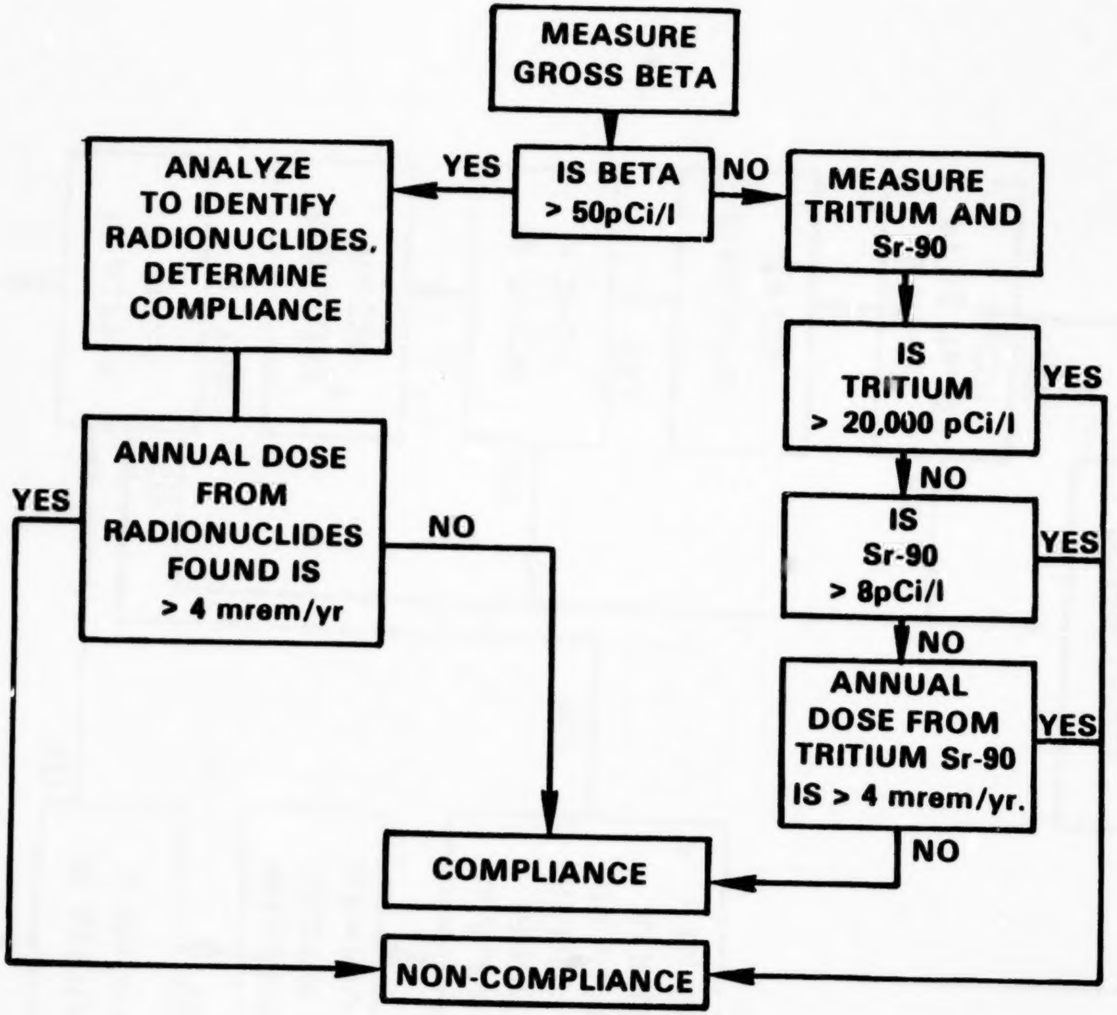
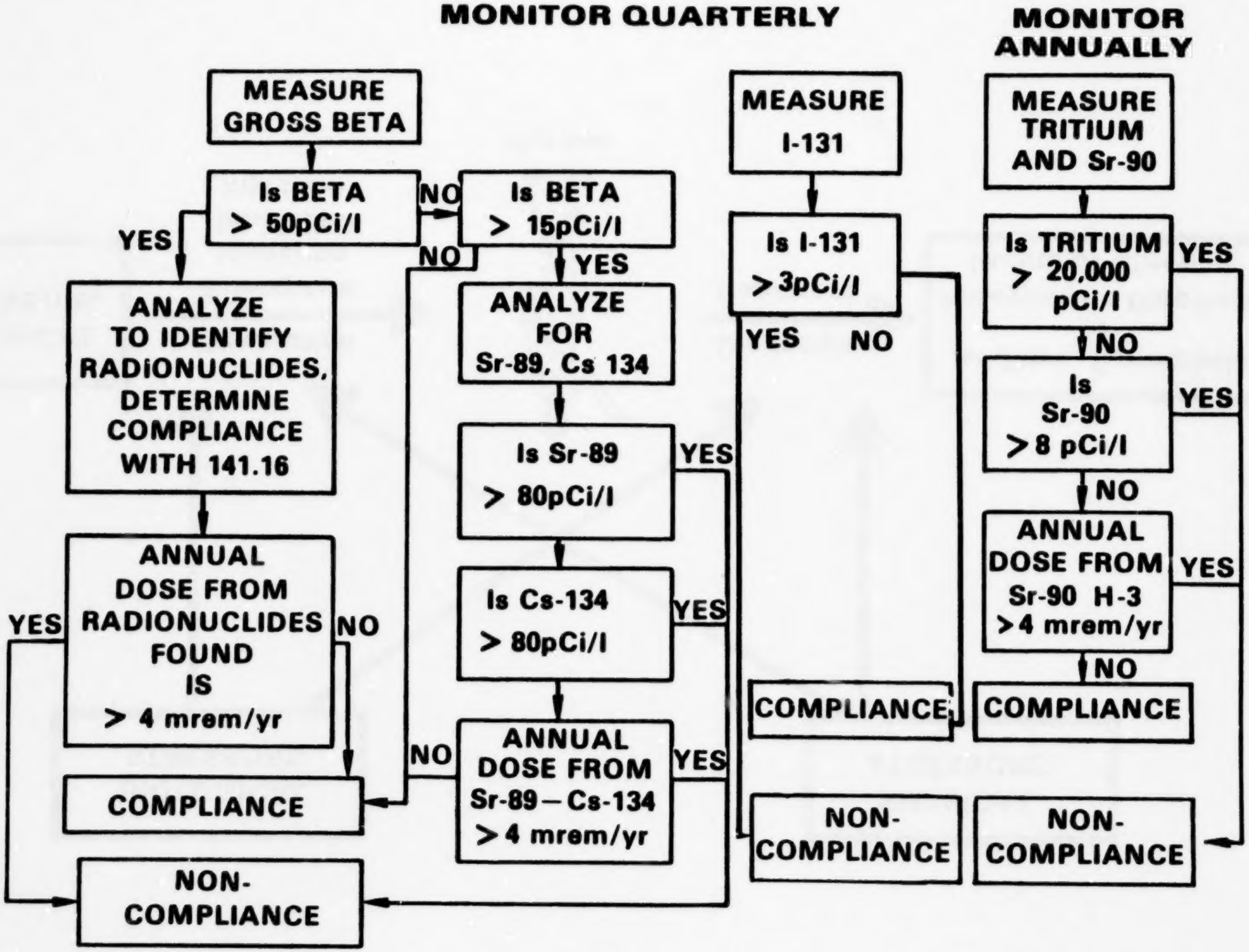

MEASURE GROSS BETA
IS BETA > 50pCi/l
YES
NO
ANALYZE TO IDENTIFY RADIONUCLIDES, DETERMINE COMPLIANCE
MEASURE TRITIUM AND Sr-90
IS TRITIUM > 20,000 pCi/l
YES
NO
IS Sr-90 > 8pCi/l
YES
NO
ANNUAL DOSE FROM TRITIUM Sr-90 IS > 4 mrem/yr.
YES
NO
ANNUAL DOSE FROM RADIONUCLIDES FOUND IS > 4 mrem/yr
YES
NO
COMPLIANCE
NON-COMPLIANCE

Figure 2


MONITOR QUARTERLY
MONITOR ANNUALLY
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YES
Is BETA > 15pCi/l
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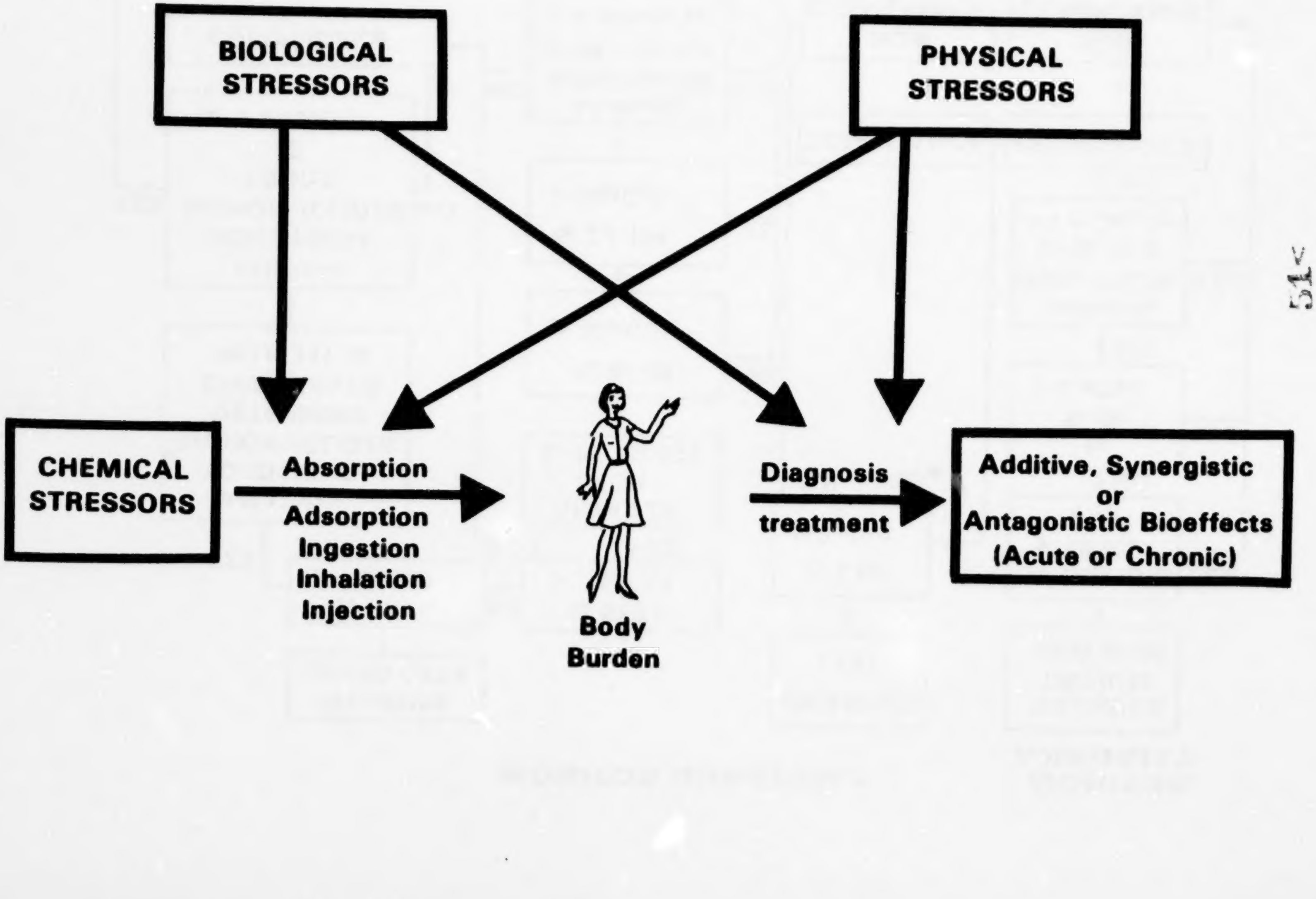
YES
ANALYZE TO IDENTIFY RADIONUCLIDES, DETERMINE COMPLIANCE WITH 141.16
ANALYZE FOR Sr-89, Cs 134
Is Sr-89 > 80pCi/l
YES
Is Cs-134 > 80pCi/l
YES
ANNUAL DOSE FROM Sr-89 – Cs-134 > 4 mrem/yr
YES
NO
ANNUAL DOSE FROM RADIONUCLIDES FOUND IS > 4 mrem/yr
YES
NO
COMPLIANCE
NON-COMPLIANCE
MEASURE I-131
Is I-131 > 3pCi/l
YES
NO
COMPLIANCE
NON-COMPLIANCE
MEASURE TRITIUM AND Sr-90
Is TRITIUM > 20,000 pCi/l
YES
NO
Is Sr-90 > 8 pCi/l
YES
NO
ANNUAL DOSE FROM Sr-90 H-3 >4 mrem/yr
YES
NO
COMPLIANCE
NON-COMPLIANCE



**BIOLOGICAL STRESSORS**

**PHYSICAL STRESSORS**

**CHEMICAL STRESSORS**

**Absorption**

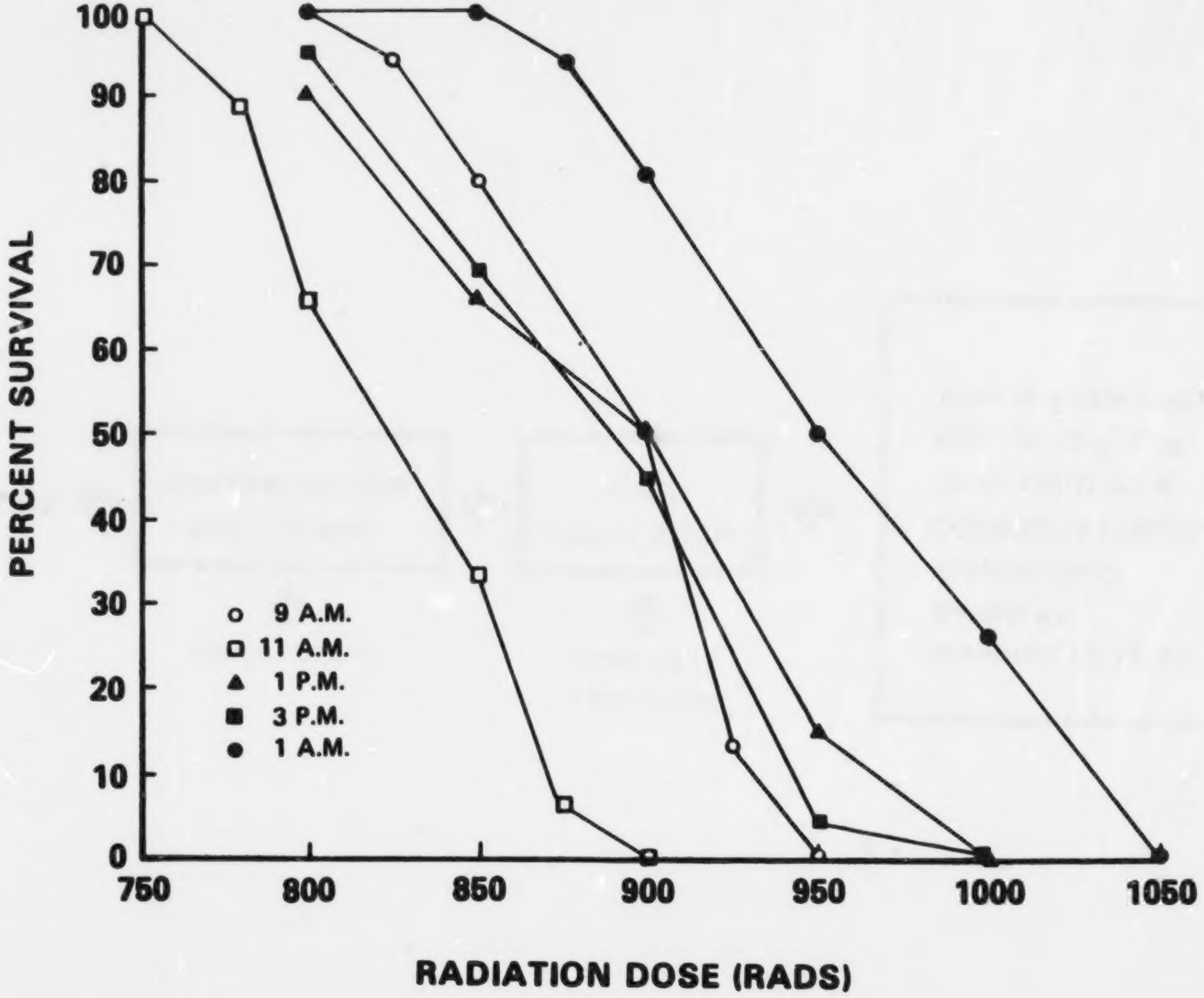
**Adsorption**
**Ingestion**
**Inhalation**
**Injection**

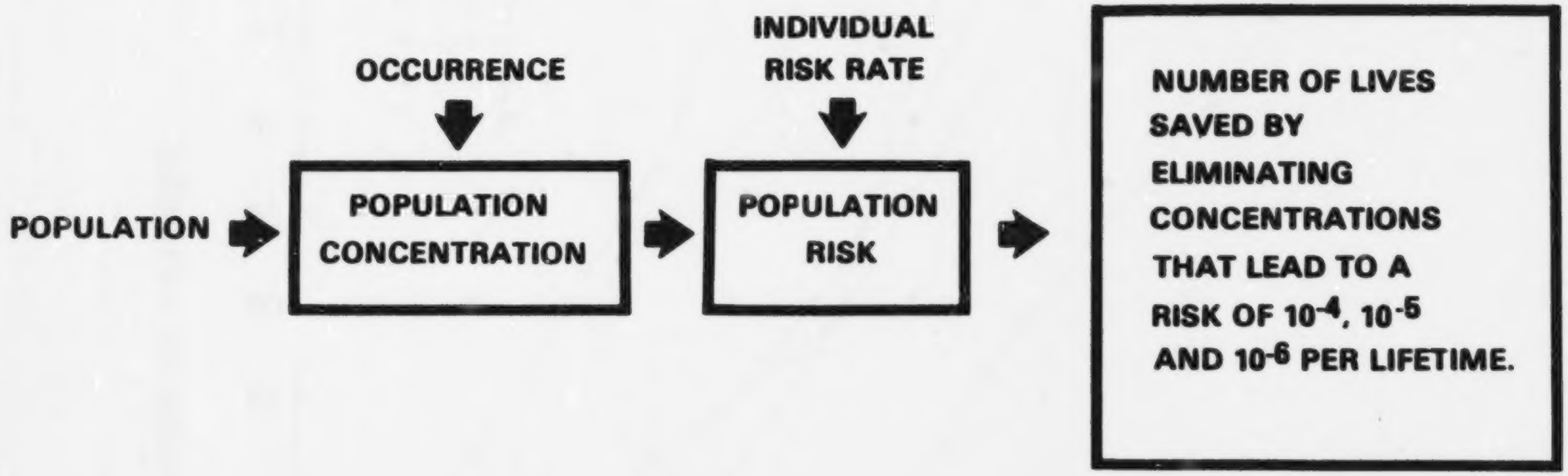
**Body Burden**

**Diagnosis**

**treatment**

**Additive, Synergistic**
**or**
**Antagonistic Bioeffects**
**(Acute or Chronic)**


PERCENT SURVIVAL
100
90
80
70
60
50
40
30
20
10
0
750
800
850
900
950
1000
1050
RADIATION DOSE (RADS)
○ 9 A.M.
□ 11 A.M.
▲ 1 P.M.
■ 3 P.M.
● 1 A.M.


OCCURRENCE
INDIVIDUAL RISK RATE
POPULATION
POPULATION CONCENTRATION
POPULATION RISK
NUMBER OF LIVES SAVED BY ELIMINATING CONCENTRATIONS THAT LEAD TO A RISK OF $10^{-4}$, $10^{-5}$ AND $10^{-6}$ PER LIFETIME.

# Table 1

## DOSE EQUIVALENT AND RISK RATE ESTIMATE FOR THE INGESTION OF URANIUM

| Isotope | Organ | Risk[a] | Dose Equivalent Rate[b] |
|---|---|---|---|
| $^{238}U$ | ENDOSTEAL BONE | 8.9 | 84 |
| | BONE MARROW | 12.0 | 5 |
| | ALL OTHERS | 10.3 | – |
| $^{234}U$ | ENDOSTEAL BONE | 11.1 | 103 |
| | BONE MARROW | 16.6 | 7 |
| | ALL OTHERS | 9.3 | – |

a = NUMBER OF PREMATURE DEATHS PER LIFETIME PER MILLION PEOPLE EXPOSED FOR INGESTION OF 20 pCi/day (50% via each isotope)

b = DOSE EQUIVALENT RATE IN mrem/yr. AT 70 YEARS FROM INGESTION OF 20 pCi/day

Source: USEPA, OFFICE OF DRINKING WATER. HEALTH ADVISORY FOR URANIUM.

# Table 2

## DOSE EQUIVALENT AND RISK RATE ESTIMATE FOR THE INGESTION OF RADIUM

| Isotope | Organ | Risk[a] | Dose Equivalent Rate[b] |
|---|---|---|---|
| $^{226}Ra$ | ENDOSTEAL | | |
| | BONE | 8.5 | 83 |
| | BONE MARROW | 30.8 | 8.6 |
| | ALL OTHERS | 5.1 | – |

a = Number of premature deaths per lifetime per million people exposed for ingestion of 10 pCi/day

b = Dose equivalent rate in mrem/yr at 70 years from ingestion of 10 pCi/day

Source: USEPA, OFFICE OF DRINKING WATER. HEALTH ADVISORY FOR URANIUM.

# REEGULATORY CONSIDERATION IN RADIATION PROTECTION

William A. Mills

# REGULATORY CONSIDERATION IN RADIATION PROTECTION

William A. Mills
U.S. Nuclear Regulatory Commission

## ABSTRACT

A regulatory scheme is suggested that identifies regions of "unacceptable" and "safe" as the upper and lower bounds and "operational" in the continuum between. These regions are associated with levels of annual risk of cancer death for a given level of lifetime exposure between 100 mrem/yr and 1 mrem/yr, upper and lower bounds, respectively. Concern is expressed with establishing public health standards at ALARA levels, which result in lower standards for reference, and views are presented on several issues of interest in regulations for protection of public from radioactivity in drinking water. Based on the regulatory scheme suggested, author concludes that existing standards for drinking water appear to be lower than need be.

## INTRODUCTION

Wise members of Congress write perfect environmental laws under which Solomon-like decisions are made by regulators to provide an environmental paradise. If you believe this statement is true then what I have to say will have little, if any, meaning to you. I will assume that most of you believe this statement is at least partially false.

In this light I would like to share with you some thoughts on regulations that I have formulated during my years of being involved in attempting to set Federal radiation protection standards. I offer these thoughts totally as my own and in no way should they be considered as views of others within the Nuclear Regulatory Commission. My comments will be long on philosophy and short on facts; this is because I believe our current philosophy on radiation protection is in need of repair. We have a wealth of scientific facts behind us as evidence by the outstanding reports of this workshop. Simply stated my request or plea, if you will, is that we apply common sense in establishing standards; an environmental paradise has never existed nor can we produce one.

Protecting public health does not mean zero risk; even for vaccines against diseases some risk is involved with the vaccine itself. I believe that the public does understand not having protection with zero risk and on a purely health basis is willing to accept some environmental risks. The question is then how much risk.

My concern is that with our desire to protect the health of our children, grandchildren, and future generations (that is provide them a very high degree of protection) we are in fact jeopardizing their economic safety. As we establish lower and lower radiation protection standards, we establish new reference points to be considered. What has happened is that "as low as reasonably achievable (ALARA)" has become not the operational and judgmental radiation protection tool that was originally intended, but in fact our standard. If it is possible to set a lower standard, do it, appears to be our current philosophy--reasonableness or common sense appears to be less in evidence. Are we than under the banner of conservative health protection running up a much heavier economic burden for our offspring?

Shown below are three principal elements ususally considered by a public health agency in meeting its responsibilities to protect health against adverse effects from a given environmental agent.

- HEALTH
- BENEFIT
- COST

The public first concerns itself with whether the contaminating agent is "safe" or "unsafe" for health. If the risk is so high that the agent is considered unhealthy by any reasonable standards, then the debate is likely to be moot and cost is considered irrelevant. The same is true, I believe, when any health risk is trival or unquestionably safe--the public does not want to see its money spent on insignificant risks.

Benefit is not generally considered in the standard setting process when the word is used in the context of "raison d'etre" of the activity producing the radiation exposure or of the source itself. Most often the benefit is a given, such as having drinking water or the need for electrical generation. Thus, for the purpose of this workshop no further discussion of this element is warranted.

Given then that the public will not accept an "unhealthy or unsafe" environment and that a benefit of the source exists, we are faced with consideration of cost. Some would argue that a cost effectiveness analysis or an evaluation of dollars spent to avoid a health detriment is a proper trade-off approach, but it too must be judgmental and rely on common sense. If we regulators were Solomons, it might be a tool, complete in itself. But we are not so wise, and we often bias our analyses and evaluations to meet our prior objectives. We have a tendency to overestimate risk and underestimate cost and those we regulate often do the reverse. So it becomes a numbers game on costs and health risks--trusting that the final position is closer to the correct one that benfits the U.S. citizenry.

Congress has not explicitly stated that the health risk from drinking water containing radioactivity must be zero and the public appears willing to accept some risk. I suggest we use these assumptions in establishing standards that are based on our judgment of what risk is safe and what risk is unsafe. We cannot give unequivocal answers to what are the risks because, as Dr. Mays (attending this workshop) stated a few years ago, "science is untidy" (Ma78), but I believe this is recognized by a public that still ranks scientists high in their confidence ratings.

In making these decisions, ICRP (1977) has provided the following guidance which can be helpful:

1. It has established a risk based system in which the health risk resulting from total body exposure is the point of reference. By weighting the risk to specific organs from internally deposited radionuclides against this reference level, total effective whole body risk from both internal and

external exposures can be summed. For drinking water, our concerns are limited only to internal emitters, and the weighting factors allow us to estimate the risk from intakes of multiple radionuclides in order to obtain an "effective total body" risk estimate.

2. It has also stated that risks to the public that are of the order of $10^{-5}$ $y^{-1}$ or $10^{-6}$ $y^{-1}$ appear to be in line with risk generally accepted in everyday living.

If we accept ICRP's guidance for a risk based system, we can propose a regulatory system for protecting the public that in general prescribes what constitutes both "safe" and "unsafe" levels of risk. Between these two bounds of "safe and "unsafe" is the range for decision in which some considerations of cost of controls and other factors (non-health) become of interest. The regulatory scheme shown below based on levels of annual risk of dying of a radiation induced cancer from continuous radiation exposure at a given associated annual dose equivalent rate depicts this point.

UNACCEPTABLE REGION
$10^{-5}$/yr (Risk Generally too High for Lifetime Exposure) 100 mrem/yr

OPERATIONAL REGION
Practice of ALARA orother judgments

$10^{-7}$/yr ______________________________ 1 mrem/yr

"SAFE" REGION
(Risk low enough to be of no regulatory concern)

The associated dose equivalent levels provided are based upon the NAS/BEIR Committee (NAS80) estimate of lifetime risk of approximately one hundred cancer deaths per $10^6$ rads from low LET radiation for continuous lifetime exposure to one rad per year (linear-quadratic dose response model). I would note that the BEIR Committee also states that it "...does not know whether dose rates of gamma or x-rays of about 100 mrads/yr are detrimental to man."

Levels about 100 mrem/yr (approximately equal to natural background) are not necessarily unacceptable if they exist for only short periods of time. For example, the limit of 500 mrem/yr for the maximum exposed individual approved by President Eisenhower in 1960 as Federal Guidance (FRC60), and reaffirmed by NCRP in 1971 (NCRP71) and by ICRP in 1977 (ICRP77) is consistent with this approach. In situations where the source of radiation remains under control or will decay with a rather short half-life (days) 500 mrem/yr is not an unreasonable limit. The risk to health would still be quite small. However, in the case of drinking water these conditions are not applicable, particularly for natural radioactivity in drinking water where the source is not under control in a practical sense and, for the most part, the nuclides have long half-lives.

The levels in the operational region do not necessarily consistitute limits. It is a region in which health is generally the more important factor near the $10^{-5}$ $yr^{-1}$ risk level and cost to reduce health risk is a consideration of increasing importance with decreasing risk. It is the region in which judgment plays the major role in decisions that involve factors other than health. It is consistent with the $10^{-5}$ to $10^{-6}$ $y^{-1}$ value recommended by ICRP and a philosophy of ALARA for public health reason. At a level of risk of $10^{-7}$ $y^{-1}$ the health risk and cost of control are no longer a consideration, the risk is insignificant.

If you accept this regulatory scheme, a generic standard for limiting radioactivity in drinking water and applicable to all radionuclides collectively is warranted. Such an effective dose equivalent limit based on health risk would convey to users of the water that the limits are established because higher risks are unacceptable (unsafe) and steps are required to reduce those risks. If the level of radioactivity in the drinking water is below the limit and in the operational region, doing something about it might be considered but would not be mandatory.

Although I believe the numerical values I have shown in this scheme are reasonable, I am not suggesting that the values be adopted per se for radioactivity in drinking water. I do believe, however, that a risk based scheme as proposed can be used to address any risk from radioactivity in drinking water generically. It would eliminate the need to have separate standards for man-made contaminants and for radium and uranium.

I would like to conclude by commenting on some very specific items of interest in considering drinking water standards.

1. Population vs. maximum individual risk. When the probability of exposure is one, or when the probability is high, as it is when using water with any radioactivity for human intake, then individual risk appears to be the reasonable criterion for protection. Protecting the individual assures protection for all. Some standard developers believe that societal risk is more important and that the population size served by the water supply is an overriding factor in protection. This idea was soundly rejected by public comments on EPA's proposed interim drinking water standards for radioactivity. The public wants equal protection in the water it consumes. Population collective dose is useful to provide a health perspective on risk but even this use should be limited in time and space.

   In determining either individual or population risk it is important to not compound uncertainties, i.e., multiply conservative values by conservative values. If prudent public health policy requires a "safety factor" or "ample margin of safety" this can be introduced at the end of the calculation, after arriving at a best estimate. Too often we maximize all assumptions on use of water, periods of such use, etc., which results in unrealistic conclusions.

2. Radon. How big a problem is exposure to radon in water? Are we convinced that lung cancer risk warrants a standard for radon? I am not so convinced to date. Nevertheless, if the plan is to establish a standard for radon in water, I suggest that consideration be given to considering the risk to the non-smoker as the basis and that realistic assumptions be made concerning exposure conditions. I specify the non-smoker for two reasons: (1) the existing confounding smoking factor in miner data has been inadequately delineated in determining risk coefficients, and (2) in applying these risk coefficients when estimating risk from exposure to radon decay products for the general population the larger lung cancer rate for smokers in the population results in overestimates of radiation

risk for the non-smoker. Consideration should be given to basing our standards on lung cancer rates for the non-smoking population only.

3. Use of Drinking Water Standards. Application of maximum contaminant limits (MCL's) established under the Safe Drinking Water Act appears to be inappropriate for application broadly to water use that does not require drinking water quality. It is especially important to keep in mind how and why the standards were developed. For example, using MCL's as standards for disposal of uranium mill waste is, in my view, a misuse because MCL's apply at the tap and not at the source. Again the tendency is to use the lowest standards available as a reference point without reexamining either the reasonableness of the standard or its basis.

In conclusion I would encourage EPA to use the wealth of information provided by the outstanding scientists contributing to this workshop and to develop more reasonable standards for drinking water. Within the regulatory scheme I have suggested, the existing standards appear to be lower than they need to be.

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# COMMITTEE ON TREATMENT, WASTE MANAGEMENT, AND COST

Chairman: George W. Reid
Recorder: Tom Sorg
Committee Members: William Brinck
Floyd Galpin
Steven Hathaway
Peter Lassovszky
Roy Reuter
Richard E. Rozelle

# TREATMENT, WASTE MANAGEMENT AND COST FOR RADIOACTIVITY IN DRINKING WATER

George W. Reid
Regents Professor/Director
Bureau of Water and Environmental Resources Research
The University of Oklahoma
202 West Boyd, Room 301
Norman, Oklahoma 73019

Peter Lassovszky
Office of Drinking Water (WH-550)
U.S. Environmental Protection Agency
Washington, D.C. 20460

AND

Steven Hathaway
Municipal Environmental Research Laboratory
U.S. Environmental Protection Agency
Cincinnati, Ohio 45268

## ABSTRACT

The processes (costs, efficiencies and reliability) used to treat drinking water to remove radioactive contaminants of concern and the disposal of wastes generated by the treatment processes are analyzed and discussed. The study was limited to uranium, radium, and radon. Initially concepts of water and waste treatment in terms of their applicability to the drinking water industry were established. The alternative processes for treatment of radium, uranium, radon, water and sludges were described and evaluated, in terms of cost, efficiency, reliability, process control and feasibility.

## INTRODUCTION

The substance of this paper can be thought of as defining a series of adjustments between the occurrence of radionuclide in water and user safety and health requirements shown in Figure 1. These adjustments, treatments from occurrence to ingestion or other exposure, are not thought of in isolation, that is selections are dependent on levels defined by both source and user committees.

In general the concern is with feasible treatment and/or management of small ground water supplies for reduction/or removal of naturally occurring radioisotopes. Though this is the primary thrust, treatment on larger scale is also important for surface waters. The primary natural isotopes selected for study are radium, uranium, and radon. Manmade isotopes and other natural isotopes, thorium, and polonium, though important, were not considered at this time. Also of concern is whether or not, and under what conditions treatment to reach a defined level is feasible. There are also questions of level and risk.

Removed waste issues addressed include RCRA and superfund, legal bases in states, sludge classification, storage dilution, isotopic dilution, gasification, thickening, ultimate disposal and recovery.

## Background

The National Interim Primary Drinking Water Regulations (NIPDWR) established maximum contaminant levels (MCLs) for natural and manmade radionuclides (Na76). The MCLs for naturally occurring radioactivity include radium 226 and radium 228 and gross alpha particle activity. The MCL for manmade radionuclide contaminants include beta and photon emitters. EPA is in the process of revising the NIPDWR, and the Agency is considering the possibility to propose MCLs for uranium and radon.

Radium, uranium and radon are naturally occurring elements and are primarily present in ground waters. The radiation problems in ground waters are attributed to the leaching of radium and uranium from rock-bearing strata. Elevated radium and uranium levels in surface waters have been attributed to mining operations. The occurrence of radium in surface and ground waters has been investigated in a number of studies (Mc60, Sc76, Ka77). Kaufmann *et al*. (Ka77) noted that the radium content of surface waters ranges from 0.01 to 0.1 pCi/L, while some ground waters may contain as much as 100 pCi/L. Drury (Dr81) estimated that of the 60,000 community drinking water supplies in the United States,

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100-2,000 would exceed a uranium concentration of 10 pCi/L and 2,500 to 5,000 would exceed 5 pCi/L. Radon is an inert noble gas and is a product of the decay of radium 226. Some amount of radon can be found in all ground waters, however, a number of studies investigating the occurrence of this element indicate large variations in its concentration (Ku54, Sm61, A175, Oc76). Duncan *et al*. (Du76) reporated that in some areas radon concentration levels in drinking waters may range between 1,000 and 30,000 pCi/L. The exposure from radon in drinking water to humans is by inhalation and ingestion. However, Partridge *et al*. (Pa79) found that airborne radon released during normal household activities such as showers, diswashers, etc., poses a greater potential health risk than radon ingested with water.

Manmade radionuclides do not occur naturally in drinking water. These radioisotopes may find their way into the water from several sources. Other than those that may result from nuclear weapon fall-out, the most likely sources of manmade radionuclides are from nuclear power plants, research and industrial facilities, radioactive storage and waste disposal sites. There may be a number of radioisotopes that may be present in manmade nuclear wastes. The composition of radionuclides depend on the type of activity the wastes originate from. In an event of a spill or fallout, the presence of manmade radionuclides will be primarily in surface waters. Leaching of contaminants from waste disposal sites would result in ground water contamination, and would eventually result in contamination of surface waters.

The naturally occurring radionuclides are alpha emitters, such as, uranium, radium, 226 and 228, and radon 222, while in general, the manmade radioisotopes are beta and gamma emitters. Ingested and inhaled radiation can be a serious health risk and in general, the absorbed dose and biological effect from alpha radiation is estimated to be twenty times more damaging than from beta and gamma radiation.

There are several different treatment methods that can be used for removing radionuclides from drinking water. It must be understoood that each radionuclide has its own specific treatment method. The property of radioactivity has little or no effect on the extent of removal by a particular treatment method. Therefore, if there are a number of different radionuclides present in the water, a specific treatment process may not be applicable to remove all of them. In those instances, multiple treatment techniques may be required.

Prior to implementing any kind of treatment, a utility should evaluate the options that are available to reduce the identified radionuclides that are present in the drinking water. The major considerations in selecting the appropriate options are technical feasibility, economics, capital and operating costs. In some instances treatment may not be the best option. A proximity of an alternate water supply with suitable water quality may make it more economical to the utility to switch to that water source instead of implementing treatment. Purchase of water from another utility may be another feasible alternative to treatment. Implementation of point-of-use treatment rather than centralized treatment may be another possibility. Nevertheless, whatever option is selected by the water supplier, it must be based upon a case-by-case technical evaluation of the system's entire process and the assessment of the economic factors involved. In selecting an appropriate treatment method, the major factors of consideration should include quality of the source water, extent of contamination by radionuclides of concern, economies of scale, ability of the community to absorb the costs, disposal of wastes produced during the treatment processes. The effectiveness of some of the treatment techniques identified below have been proven in actual practice. The other treatment technologies which are discussed have been evaluated in the laboratory and pilot plant studies. The result of these studies indicate that these technologies may be potentially viable methods to remove radionuclides from drinking water.

## GENERAL CONCERNS ABOUT TREATMENT TECHNOLOGIES

Treatment technologies are derived from water works practice, and perhaps will affect not more than 600 of some 60,000 public water supply systems in the U.S.A. The degree of removal of a specific contaminant from source waters is limited by the efficiency of the particular treatment applied. Treatment of source waters containing high concentration levels of contaminants will produce poorer quality of processed waters compared to waters containing lower levels of contaminants prior to treatment. This is illustrated in Table 1. Treatment can be thought of as a tree diagram, in which, one process is layered on another or on itself to achieve increasing efficiencies. It is basically a problem of solid concentration, such that the cost to remove from 80% to 90% efficiency is the same as from 90% to 95%, as 95% to 97.5%, etc., that is the solids are doubled from 97.5% to 95% as from 95% to 90%, etc. So if one can achieve 90% efficiency, the next 5% will cost him as much as the previous efficiencies, to go to 97.5% would be another increment. Where high source concentration of contaminants exist or the maximum allowable level of contaminant iin the product water is very low, it may be necessary to use several stages of treatment. In that case the cost of producing acceptable water multiplies, and other options, dilution, splitting, blending, point-of-use treatment, etc., become important. There are also unanswered questions as to treatment induced conflicts, such as sodium discharge from ion exchange columns and heart disease, chlorine for disinfection, radioactivity and cancer, etc.

## Radium Removal Processes

Processes that are effective in removing radium from drinking water include lime softening, cation exchange, reverse osmosis and selective absorption. The efficiencies of these processes have been domonstrated by full scale operating facilities, pilot plants and laboratory studies. (See Table 2)

## Cation Exchange

Ion exchange using natural or synthetic resins to replace calcium and magnesium ions with sodium ions to soften the water is a widely practiced technology. If radium is present in the water it will be removed with the hardness since radium is a divalent cation and similar in chemistry to calcium and magnesium. As illustrated in Table 3, experience in Iowa and Illinois indicates that a well operated ion exchange softening plant is capable of removing 85-97% of the radium from drinking waters (Be76, Br78, Sc76). Since radium removal still takes place after the resin is exhausted for hardness, regeneration of the resin to achieve good hardness removal will assure good radium removal also (Ep77).

There are several advantages and disadvantages to the use of ion exchange softening systems. One major advantage is that ion exchange is readily adaptable for small systems. The units are commonly available, relatively simple and easy to operate. In addition, ion exchange systems require relatively small space. In areas where the water is hard, ion exchange will perform a dual purpose. It will remove hardness along with the radium. However, an area of concern associated with the use of sodium ion exchange softeners is that as a result of treatment the sodium content of the water increases. The National Academy of Sciences (Na77) concluded that in areas where the sodium content of the water is already high, this may be a potential problem for people with restricted diets and hypertension. Ciccone (Ci83) noted that in order to avoid the addition of sodium, potassium chloride may be substituted as a regenerant. However, potassium chloride may be as much as five times more expensive than sodium chloride. Another alternative to sodium cation exchange is hydrogen ion exchange to remove radium from drinking water is being investigated at the University of Illinois (Cr80). Sorg (So81) notes that potential saving with this process may be realized if sulfuric acid is used as a regenerant since the stoichiometric amount of sulfuric acid required for regeneration of the resin is considerably less than the sodium chloride required for regenerating a sodium ion exchange system. On the other hand, the hazards associated with the handling of the acid used for regeneration may preclude the use of hydrogen exchange resins for home or point-of-use applications.

Another disadvantage associated with the use of softeners is that the softened water can be corrosive to the distribution system. In some instances the radium content of the untreated water may be low enough to allow blending with the treated water, thus producing a less corrosive water. Where the radium content of the untreated water is excessively high, treated water and unheated water cannot be blended. The treated water in this instance may require the addition of corrosion inhibitors. Although large ion exchange softening systems are technically feasible, the economies of scale make them less attractive compared to lime softening. Most of the larger water supply systems use surface waters that may require pretreatment such as filitration before ion exchange could be used.

## Reverse Osmosis

Reverse Osmosis (RO), a relatively new technology is being commonly used in areas where the water has high total dissolved solids concentration levels (TDS). The RO process utilizes a membrane which allows the passage of the water but rejects the dissolved salts. Only a portion of the water is treated. Pressure is required to force the water through the membrane. The removal efficiency of RO is better at higher pressures. The process is continuous and the rejected concentrate is discharged as a continuous stream. Experience in Florida by Sorg (So80) and Iowa by Bennet (Be76) indicates that RO is highly effective in removing radium from drinking water. As shown in Table 4, the process can be over 90% efficient.

The economic advantages are most favorable when using RO for the removal of radium if the drinking water contains other contaminants to be removed at the same time. Space requirements for RO are small compared to equivalent capacity lime softening or ion exchange systems. Sorg (So80) notes that RO units for small systems are prefabricated and pretested at the factory so their installation can be completed in a short time.

Compared to other treatment techniques RO is relatively expensive to operate due to high energy requirements for pressure pumps. In addition, where feed waters contain suspended solids, iron, manganese and scale-forming compounds, pretreatment (lowering of the pH) of the water is required before RO can be used. Since RO also removes the hardness from the water, stabilization of the water may be required to make it non-corrosive.

## Lime Softening

Lime softening is a commonly used process to remove hardness from the water. Lime is added to the water to neutralize the carbonic acid and to form insoluble calcium carbonate and magnesium hydroxide which will precipitate out during the removal process. Experience in the field and the laboratory (Br78, EP77) shows that lime softening can remove 80%-90% of the radium provided the process pH is maintained above 10. The results of field studies are shown in Table 5.

Although small lime softening systems may be technically feasible, this treatment technology is best suited for large capacity plants. Lime softening systems require more complicated equipment and demand more operating supervision. Lime softening is also well suited to the treatment of waters containing iron and turbidity since these contaminants are also reduced.

## Adsorption Processes

Adsorption processes for radium removal are in the development stage with the exception of greensand filtration which has been used extensively to remove iron and manganese from water. However, because of their potential for removal of radium, these technologies should be given consideration as potentially viable technologies. One of the adsorption processes utilizes the capability of manganese dioxide ($MnO_2$) to adsorb metal ions, while the other process involves a radium selective complexer developed by DOW Chemical Corporation.

$MnO_2$ Adsorption. Raw water containing iron and manganese can be treated by passing it through greensand filters after oxidation. It has been observed in Iowa and Illinois (Ep77) that potassium permanganate added to the water as an oxidant, improves radium removal efficiency. However, as shown in Table 6, the radium removal efficiency is limited to not more than 56%. The removal of the radium is attributed to the manganese dioxide ($MnO_2$) formed during the oxidation. The $MnO_2$ is known to be an effective adsorber of many metal ions. In order to increase the efficiency of the process and to determine its technical and economic feasibility, EPA has funded additional research to conduct pilot and field studies in Iowa (Rc81).

Because of low radium removal efficiency, the use of iron removing greensand filters is limited to instances where radium levels do not exceed twice the MCL for radium. However, when this technology is used, no sodium is added to the water as a result of treatment. Iron, manganese, and radium are all removed by this method.

Additional field studies by Cook (Co68) indicate that manganese dioxide impregnated fibers may remove radium more effectively than greensand filters continuously fed with potassium permanganate.

The treatment consists of passing water through a filter vessel loaded with manganese dioxide impregnated fiber media. Once the fiber media is exhausted, it is replaced with a new one. Since the preparation of the fibers is not simple, it may pose operational problems in small system application where no qualified operators are available. No backwashing or regenerating of the system is required, thus eliminating the need for disposing the wastewater discharge. Results of the studies by Cook (Co68) indicate that this process may be able to remove radium from

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relatively large amounts of water. However, the practical performance of the units may be adversely affected by the washout of loosely held manganese oxide from the fiber.

DOW Radium Selective Complexer (RSC). This material was originally developed by DOW Chemical Company to remove radium selectively from minewater effluents. The RSC system is basically a water softener unit in which the normal exchange media is replaced with the RSC. Boyce (Bo82) reported that the RSC has a very high loading capacity for radium. Initial field test data indicated that the RSC reduced radium levels ranging from 20 to 45 pCi/L in the water to below 3 pCi/L for approximately four months without any need for regeneration. Similar results were obtained in Brady and Bellville, Texas, where that the RSC was capable of reducing radium levels from 30 pCi/L to below 5 pCi/L without any need for regeneration (Te82). The potential for the RSC as an effective treatment method to control radium in drinking water is considerable. Due to its simplicity the process could be adopted to both small and large water supply systems which need to treat the water for radium removal. Because of the RSC's high loading capacity for radium the sorbent units may be kept in service for a long time before replacing the sorbent media. The operational requirements for backwashing and regenerating the units would be eliminated as well as the problems associated with the disposal of waste streams. Since hardness is not removed during the process, post treatment for corrosion control would also be eliminated.

However, by eliminating the need for regeneration, the buildup of radium in the RSC may be a potential concern. Therefore, additional studies are needed to evaluate the possible hazards associated with the radiation fields resulting from the accumulation of radium in the adsorbent bed.

## Direct Precipitation by a Soluble Barium Compound

Kerr-McGee Corporation commenced in 1973 examining methods of radium removal from a by-product stream in order to permit its use directly as a fertilizer solution (Sh83). Regulations subsequently were established for point source discharges of uranium minewater requiring the control of soluble radium-226 at a level of 3 pCi/L on the average with a maximum of 10 pCi/L. Kerr-McGee uranium mines, expected to be operating in the 1980's, could be expected to discharge approximately 10,000 gallons per minute cumulatively. It became imperative to determine technology which permitted the reliable control of radium to these established levels. As a result of work previously done with radium solutions in AEC processes and a classical analytical procedure for the removal of radium, direct addition of a soluble barium solution to process by-product and minewater effluents, containing excess sulfate ion, promised the possibility of radium control at the desired levels. Over a period of time, Kerr-McGee evolved a direct precipitation procedure employing soluble barium compounds, i.e., barium chloride, carbonate, nitrate, which when dissolved in water at the appropriate concentration, could be added to a radium containing solution of 50 to 200 pCi/L by a metering pump at the appropriate rate. This addition at the metered rate resulted in the precipitation of radium as a barium-radium sulfate precipitate which is then permitted to grow in the fashion postulated by Ostwald, resulting in particles of sufficient size to be removed by simple settling.

The design of the settling ponds was determined empirically to be approximately as follows. The pond would be designed with a length-to-width ratio of approximately 4:1, inflow and outflow controlled to secure approximately 1 foot per minute velocity through the pond between inlet and outlet with a total residence time of 72 hours. Measurements of the pond's efficiency normally disclosed that the efficiency of pond in achieving these conditions was in the range of 70% to 80%. Thereby, providing a true residence time of approximately 50 hours. This design provided the ability to reduce radium contents to required levels or below. Radium concentrations on the order of 1 pCi/L or less have been routinely maintained by treatment systems of this type.

This technique of radium control has been successfully applied on an experimental basis to solutions containing 60,000 pCi/L successfully reducing terminal concentration to less than 5 pCi/L.

Probably this technique could be applied to a drinking water supply at a cost somewhat less than current water treatment techniques such as reverse osmosis, ion exchange, etc., as reported in the current literature.

The technique does require careful control of barium concentrations injected into the water and routine analysis of of the treated water to assure achievement of target concentrations.

## Uranium Removal Methods

Due to the absence of standards limiting the presence of uranium in drinking waters, development of treatment techniques to remove uranium from water has been aimed at recovery operations from mine process waters and effluents. However, recent studies by Drury (Dr82) and Bondietti, White and Lee (Le82) at the Oak Ridge National Laboratory (ORNL) and by Hathaway (Ha82) indicate that a number of treatment techniques have the potential to reduce and maintain uranium levels in drinking waters at or below 10 pCi/L. These treatment techniques include anion exchange, lime softening, reverse osmosis and under certain conditions conventional coagulation using alum or iron salts.

## Anion Exchange

Because of their effectiveness and relatively low operating costs, anion exchange resins have been used to recover uranium from mine process waters. Laboratory and field studies by Bondiette, White, Lee (Le82), and Hathaway (Ha82) indicate that anion exchange technology is feasibile to remove uranium from drinking waters. The results of laboratory studies by Bondietti, White and Lee (Le82) (Figure 2), indicate that anion exchange resins have a large adsorption capacity and high selectivity for uranium. Bench scale studies at EPA by Hathaway (Ha82) (Figure 3) show that when using actual drinking water contaminated with 300 microgram uranium/L, over 9,000 bed volumes were treated before uranium was detected in the effluent. Field studies by EPA confirm the findings of these laboratory studies. These studies involved the evaluation of the performance of twelve 1/4 cubic foot anion exchange systems installed in New Mexico, Colorado, and Arizona at sites where the uranium levels in the untreated water were in excess of 10 micrograms/liter. The preliminary results of these field studies are shown in Table 7. Because of the high loading capacity of the anion resins for uranium, these units are especially suitable for point-of-use applications where on-site regeneration is not feasible. For centralized treatment, the resin may be regenerated and recycled by backwashing it with sodium chloride solution.

## Activated Alumina

The EPA has also evaluated activated alumina as a removal media for uranium. Small columns packed with Alcoa activated alumina (129 mL) were used to treat raw water containing

300 micrograms uranium/liter. Figure 4 shows the general removal efficiency found in the laboratory study. Subsequent regeneration and treatment revealed that approximately 5,000 bed volumes could be treated to total exhaustion. No pretreatment of the raw water was practiced in this study.

## Lime Softening

Laboratory studies by Bondietti, White, and Lee (Le82) indicate that lime softening may be effective in removing uranium only if the pH of the water is maintained between 10.6 and 11.5. At these pH levels 85% to 90% removal of uranium may be achieved as shown in Table 7. The removal efficiency of uranium may be improved to 99% by the addition of 80 to 120 ppm magnesium carbonate to the water, provided the lime doseages are maintained at or above 100 mg/l as illustrated in Table 8. At less than the above dosages of lime and magnesium carbonate, the uranium removal efficiency will be reduced drastically. Use of modified lime softening techniques (magnesium addition) to remove uranium is especially suitable in communities where lime softening is already used to treat the water.

## Conventional Coagulation Techniques

Limited information is available regarding the effectiveness of coagulation techniques to remove uranium from drinking waters. Laboratory studies by Bondietti, White and Lee (Le82) evaluating the effectiveness of iron salts and alum indicated that uranium removal efficiencies are very sensitive to pH. The removal efficiency at a specific pH level depends on the prevailing charge on the floc and the uranium species present. Table 9 compares the charges of alum and ferric salt flocs with the suspected uranyl species present in the water at various pH levels. Because of the unstable nature and solubility of alum, coagulation may be the most effective at a pH level of 6 as shown in Table 10. Iron salts may be used at a wider pH range because they are more stable. Use of coagulant aids such as polymers may improve the efficiency of treatment and may reduce the alum dosage requirements. Practical plant operating data reported from Arvada, Colorado, for the year 1982 shows that raw water uranium levels varied from 24 micrograms/liter to below detectable levels depending on season. Effluent uranium was always 3 micrograms/liter or less. The treatment method was coagulation with 12 - 25 mg/L alum, 0.2 mg/L polymer, and direct filtration of the coagulated water. Because of limited practical experience dealing with the application of coagulation technology to remove uranium from drinking waters, additional studies are needed to determine the practicality of this technology. However, in instances where coagulation is already used for water treatment, and uranium removal from the water is desirable, the utility should investigate the possibility of modifying the process before adding on a different

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treatment process. The selection of appropriate coagulant or coagulant aid, the determination of actual dosages and pH adjustment must be done through onsite laboratory and pilot plant testing with actual raw water. In general, coagulation treatment is more suitable for larger systems where trained operators are available. However, pre-fabricated package treatment plants requiring limited operator attention are available for smaller communities.

## Reverse Osmosis

There is no practical experience involving the utilization of RO technology to remove uranium from drinking waters. However, studies evaluating RO performance in removing uranium from minewaters solutions (Sa76) indicate that RO can be over 90% effective. Results of limited bench scale studies by Bondietti, White and Lee (Le82) seem to verify these results. Since RO is effective in removing most inorganic contaminants from water it can be expected that RO may be a practical option as a treatment method to remove uranium from drinking waters.

## Electrodialysis (ED)

Another through membrane process, ED, modified by Ionic Inc., to create polarity reversing ED is competitive with RO (Re83). Studies at the Teton-Nedco Leuenberger Research and Pilot in Casper, Wyoming, by Garling, indicate comparative cost for O&M, and pretreatment. The unit operated 86% of the time, at $1.02/1000 gallons (See Table 11) plant capacity was 30,000 gallons per day.

## Radon Removal Methods

There are two methods available that may effective in removing radon from drinking water. These methods are adsorbtion by granular activated carcon (GAC) and aeration.

## Granular Activated Carbon

The effectiveness of GAC as an adsorbent of noble gases is well known. GAC has been used to remove radon from water and to concentrate it for analytical measurement. However, with the exception of studies performed at the University of Maine, there is no documented evidence of utilizing GAC as a continuous process to remove radon from drinking waters (Lo82). Because of its short half life of 3.82 days it has been found that large portions of the adsorbed radon decay within the GAC bed before breakthrough. Thus, in effect the GAC bed in the process acts as a storage vessel for the radon. Because of the decay, effective life of the GAC bed is extended many times over the life indicated by the adsorption isotherm. The removal efficiency of radon from the water is governed by the design of the GAC bed. It has been estimated that a 1.0 to 2.5 cubic feet GAC contactor is capable of removing 96% of the radon supplied to a single family home unit. Because of the

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decay of radon and radon daughters accumulated within the GAC contactor, concern has been expressed about gamma radiation hazard. Another potential concern regarding the use of GAC unit is the fate of radon daughters in the GAC bed. However, preliminary tests with a commercial unit indicate that even after four months of operation no radon daughters were detected in the effluent water. Due to their simplicity, use of GAC units to remove radon in small water supply systems are attractive. No moving parts are needed to operate the units. In areas where turbidity is present, occasional backwasing of the GAC units may be required.

## Aeration

Laboratory and field studies performed at the University of Maine by Lowry (Lo82) indicate that a well designed 35 to 40 gallon diffused aeration tank is capable of removing more than 95% of the radon. A unit of this size is suitable to meet the needs of a single family home. Hinckley (Hi82) reported that evaluation of spray aeration systems by the State of Maine indicates that 93% of the radon may be removed from the water. In general, spray aeration to remove radon from water is best suited for larger water supply systems where the radon levels are high. When utilizing aeration techniques to remove radon from water, care should be exercised to assure that humans are not exposed to the radon released into the air.

## Disposal of Water Treatment Residuals

An important consideration in determining the appropriate method of water treatment for radionuclide removal, and the associated costs, is the disposal of the treatment residuals. This appears to be a more significant potential problem in the case of small groundwater supplies remote from surface streams. Table 15 summarizes the methods of disposal of treatment wastes as identifie in the EPA 1977, "Manual of Treatment Techniques for Meeting the Interim Primary Drinking Water Regulation," EPA-600/877-005 (Ep77). These methods are considered to also be generally applicable for the disposal of wastes generated in processes used for radionuclide removal. However, there are a number of special considerations that deserve specific mention.

### A. Residuals from Radium Treatment

### 1. Dried Sludges and Other Solids

Present data indicates that sludges resulting from lime softening processes and coagulation methods are not expected to exceed 5 to 10 picocuries per gram of sludge. This should not be a problem in the usual methods of sludge disposal. However, for exceptional cases that may fall outside this range several cautions are appropriate.

Sludges containing radium concentrations of greater than 5 picocuries per gram, in large volumes, must be restricted in waste disposal practices where they could be incorporated in building materials or where they would serve as the main constituent of landfill when future construction on the site is a possibility. Such restrictions will limit the possibility of excessive radon emanation into living spaces. Radium concentrations up to 100 picocuries per gram could probably be suitably disposed of in sanitary landfills where they are relatively minor constituents of the total waste in the facility. Higher concentrations of radium should be considered for designated hazardous or radioactive waste disposal sites. However, wastes of this concentration probably would not result from present conventional water treatment processes used for radium removal. Other processes now in the developmental stage, such as manganese fiber filters, may, however, produce higher specific activity residuals.

### 2. Ion Exchange

Backwashes of cationic exchange resins used for radium removal can usually be disposed of by discharge to surface streams if there is sufficient dilution to handle the salinity. In most cases special consideration of the radium content should not be necessary but may require special permit authorization. It is noted that the public perception that it is a radioactive discharge may also create problems.

## Reliability of Treatment Methods

From existing information, Reid and Law (Re83) estimated the reliability of the processes being used to remove Ra U, and Rn. This information is presented in Tables (12, 13, and 14). Reasonably sufficient data exists only on coagulation, softening, EDR and RO which indicate that these processes are more reliable.

## Costs Associated with the Removal of Radionuclides

The capital, operating and maintenance costs (O&M), and the costs for producing 1,000 gallons of water by various treatment techniques versus the size of treatment facility are shown in Figures 5, 6, and 7. The treatment techniques considered include anion exchange, cation exchange, reverse osmosis and conventional coagulation techniques using alum as a coagulant. Also included are costs for modifying lime softening and alum coagulation. In instances where one of these processes are already in use, modification of the existing process to remove radionuclides may be the most economical solution. Costs associated with the use of manganese dioxide fibers and the RSC sorbent are not illustrated in the Figures due to the lack of sufficient data. The cost of removing uranium by anion exchange cannot be represented by Figures 5, 6, and 7. The high capacity of strong base anion resins to remove uranium would substantially change the cost curves due to less frequent regeneration and low operating cost. No actual costs have been developed for this process. These costs were developed for new systems designed for capacities in excess of one million gallons per day (MGD). They were determined on the basis of work developed by the Municipal Environmental Research Laboratory, U.S. Environmental Protection Agency, Cincinnati, Ohio, using the computer model written for EPA by Gumerman et al. (GU79). The costs for new systems having capacities less than 1 MGD were developed by V.J. Ciccone and Associates in coordination with EPA and several engineering, design and construction firms. The capital costs were based upon an extrapolated Engineering News Record average construction cost index for 20 U.S. cities. Operating costs were based upon a Producer Price Index for 1982 dollars. For the purpose of estimating costs per 1,000 gallons of water treated, the capital costs were amortized over 20 years at 12% interst rate. Figures 5, 6, and 7 represent average estimated costs. The actual costs involving particular treatment facilities may greatly vary due to site specific conditions such as water quality, geographical and local conditions and waste disposal requirements. Costs for sorbents to remove radium are not included.

In some instances point-of-use treatment to remove radionuclides may be more economical than centralized treatment. The costs per customer may be significantly lower for small communities since only a portion of the water intended for consumption may require treatment. For ion exchange, small water softeners may be adopted. Home softeners are frequently purchased or leased to soften hard water. Ciccone (Ci83) estimated that a commonly used home softener system amy cost $829.00 per unit. These units may be utilized to remove radium. If regeneration at site is practical, the operational cost for salt usage may be $5.00 per month or $60.00 per year. Cartidge size units for ion exchange cost about $450.00. Replacement costs for cartridges including labor is estimated to be $138.00 per year. Removal of uranium in a home system may be considerably less expensive than removing radium. It is estimated that a 1/4 cubic foot unit could treat 20,000 gallons of uranium contaminated water. A 1/4 cubic foot unit with strong base resin would cost about $200.

Similar cartridge size units for RO are available for point-of-use. The average cost of a small RO unit for point-of-use is estimated to be $780.00. Operating costs for an RO unit include electricity and labor to replace the membrane element once per year. Replacement cost for the cartridge element including labor is estimated to be $146.00 per year. Costs to remove radon from drinking water in small household application have been determined by Lowry (Lo82). Comparison of costs to remove low, medium and high levels of radon to produce 200 gallons of water per day are shown in Table 16.

## INFORMATION NEEDS

### A. Treatment Technology

General information gaps, where further research, and data gathering needed to evaluate practical applications of treatment technologies to remove radionuclides from drinking water have been identified by the committee. These are listed below:

1. Information is needed on the operation and costs of the magnesium dioxide impregnated fiber and the barium chloride coagulation methods for radium removal.

2. Information is needed on full-scale operation and costs of electrodialysis and radium selective complexer (RSC) for radium removal.

3. Information is needed on full-scale operation and costs of anion exchange, lime softening, reverse osmosis, electrodialysis and conventional coagulation for uranium removal.

4. Information is needed on full-scale operation and costs involving GAC and aeration treatment for radon removal.

5. Information is needed on direct radiation from treatment processes and devices used for radium and radon removal.

6. Information is needed on reliability of processes in use.
7. It would be advisable for EPA to develop a standard methodology for considering calculating and integrating in an objective

manner the aspects of treatment cost, treatment performance parameters (reliability, operator skill, etc.), feasibility and availability of processes that are used for recommending all MCL's balancing the reasonableness of cost and availability of treatment against the health risks and address, or encourage others to fill in obvious gaps of information.

B. Waste Disposal

General information gaps, where further research and data gathering are needed to evaluate potential waste disposal problems have been identified by the committee. These are listed below:

1. Methods for disposal of ion exchange recharge brines, where an adequate dilution surface stream does not exist, need to be developed. These may include various forms of evaporation or other means of concentration, with subsequent disposal of solid residuals.

2. There is a lack of information on the specific activities (i.e., concentrations) of uranium that may occur in the residuals from uranium removal systems. The backwash from anion exchange resins is a particular case where more detailed data is necessary.

3. A primary method of choice for the removal of radon from water appears to be aeration. The potential for creating a source of radon emanation at a water treatment facility which in turn may require control under the Clean Air Act needs to be examined.

The problem of waste disposal for small systems may be especially acute. A large number of small communities discharge their sewage into individual septic tanks. These tanks may not be able to handle the high salinity wastes generated by ion exchange units. In order to solve the waste disposal problem, the community may elect to use selective sorbents or high loading capacity resins. Upon exhaustion, the sorbent or ion exchange media may be removed from the contractors and disposed as a solid waste. In some instances the resin may be regenerated at a central site. This method of treatment and disposal is especially suited for point-of-use application.

Because of the accumulated radionuclides in the sorbent or the resin, special care or procedures may have to be used in handling and disposal. It has been shown that gamma radiation in the surface of GAC bed used to remove radon may reach levels as high as 16 millirem/hours. Thus, before disposal the GAC bed should be left idle to allow most of the radon to decay. It has been estimated that after 12 days 90% of the radon will decay. Waste disposal requirements and limitations may considerably influence the feasibility and costs associated with the overall treatment. Therefore, the utility should evaluate thoroughly the available means of waste disposal associated with particular treatment methods.

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Table 1

Boundary Levels, Treatment, Source, User

| Element | Raw (pCi/L) | Finish (pCi/L) | Percent Reduction by Treatment |
|---|---|---|---|
| Radium | 200 | 20 | |
| | 150 | 10 | |
| | 50 | 5 | 90 |
| Uranium | 200 | 100 | |
| | 100 | 40 | |
| | 50 | 10 | 95 |
| Radon | 750,000 | 20,000 | |
| | 100,000 | 10,000 | |
| | 10,000 | 5,000 | 95 |

| | |
|---|---|
| Landfill | 5 pCi/gm up to 100 pCi/gm (landfill) (under RCRA) |
| Land Disposal | 2#/acre/ (based on Cd) |
| Ocean Disposal | Trace Level Requirement |

Table 2

Summary of Process, Levels of Concentration of Removal

| Process | Removal Efficiency | Effect of Plant Size | Problems |
|---|---|---|---|
| Ion-exchange | 95% Ra | Ion exchange is generally used in batch process. Small plants are more cost effective. The available cost data only covers plant capacity up to 10 MGD. | Treated water shows an actual increase in total dissolved solids.<br>Sodium concentrations may be elevated.<br>Raw water requires pretreatment if turbidity and suspendment if turbidity and suspended solids, iron and manganese, or bacterial slimes are present<br>Treated water may be corrosive<br>Disposal of spent brines can be a serious problem.<br>Capital cost is substantial. |
| Anion Exchange | 99% U | Experimental Stage | |
| Reverse Osmosis | 95% Ra | This process is most suitable for automated plant operation and use in small plants. | High capital and operating cost.<br>Considerable pretreatment requirements particularly if raw water contains suspended solids, organic material, or dissolved gases.<br>Reject stream requires disposal.<br>Treated water must be stabilized. |

Table 2

Summary of Process, Level of Concentration of Removal (Continued)

| Process | Removal Efficiency | Effect of Plant Size | Problems |
|---|---|---|---|
| Lime and Lime-Soda Softening | 80-90% Ra | Best for large plant capacity over 10 MGD<br>This process is the Choice for large treatment plants. | Process is more difficult to control than ion-exchange or reverse osmosis.<br>Operating costs are high, particularly chemical costs.<br>System size limitation.<br>Requires significant operation attention.<br>If hardness is not a problem in the water the lime-soda softening process is not cost-effective to remove just radium |
| Greensand | 50% Ra | | Water having less than 10 pCi/L radium may be treated with this technique.<br>Requires disposal of waste material. |
| Mn-Fiber | 90% Ra | Testing stage laboratory scale | Manganese oxides are not completely bound to the fiber and up to 50% may be washed off.<br>The fiber is difficult to prepare and handle. |

Table 3

RADIUM REMOVAL IN ION EXCHANGE PLANTS

| Plant | Ra in pCi/L | Ra out pCi/L | %Ra Removed | Hardness in mg/lCaCO$_3$ | Hardness out mg/lCaCo$_3$ | Hardness % Removed |
|---|---|---|---|---|---|---|
| Eldon, IA | 49 | 1.9 | 96 | 375 | 10 | 97 |
| Estherville, IA | 5.7 | 0.3 | 95 | 915 | 46 | 95 |
| Grinnell, IA | 6.7 | 0.2 | 97 | 385 | 11 | 97 |
| Holstein, IA | 12 | 0.5 | 96 | 920 | 18 | 98 |
| Dwight Corr. Inst., IL | 3.26 | 0.36 | 89 | 286 | 43 | 85* |
| Hercher, IL | 14.31 | 1.31 | 91 | 401 | 60 | 85† |
| Lynwood, IL | 14.69 | 0.41 | 97 | 848 | 78 | 91 |

* Removed

† Hardness and %Ra removals are somewhat low due to breakthrough occurring prior to all samples being collected.

Source: Bennet, D.L, *et al.* (1976), Schliekelman, R.J. (1976), Brinck, W.L., *et al.* (1978)

## Table 4

### Radium Removal in Reverse Osmosis Plants

| Plants | Ra in pCi/L | Ra out PCi/L | % Ra Rem. |
|---|---|---|---|
| Greenfield, IA | 14.0 | 0.6 | 96 |
| Sarasota, FL | 22.0 | 0.8 | 96 |
| Bay Lakes Estates | 3.2 | 0.14 | 95.6 |
| King Gate | 15.4 | 2.0 | 87 |
| Spanish Lakes | 10.5 | 1.2 | 88.5 |
| Sorrento Shores | 4.6 | 0.21 | 95.4 |
| Venice | 3.4 | 0.6 | 82.4 |
| Bay Front | 12.1 | 0.6 | 95 |

Source: Sorg, T.J. (1980), Bennet, D.L. et al. (1976).

## Table 5

## Radium Removal in Lime Softening Plants

| Location | Ra In pCi/L | Ra Out pCi/L | % Ra Removed | pH |
|---|---|---|---|---|
| Des Moines, Iowa | 9.3 | 2.35 | 75 | 10.4 |
| Webster City, Iowa | 6.1-7.8 | 0.3-0.9 | 85-96 | 10.05-10.95 |
| Peru, Illinois | 5.48-6.49 | 0.51-1.62 | 70-92 | 8.2 |
| Elgin, Illinois | 3.51-7.45 | 0.71-0.80 | 80-90 | 10.2 |
| Venice, Florida | 8.73 | 2.19 | 75 | 9.7 |
| Englewood, Florida | 1.69 | 0.69 | 59 | 8.5 |

Source: Bennet, D.C. et al. (1976), Schliekelman, R.J. (1976), Brinck, W.L. et al. (1978).

## Table 6

### Ra-226, Iron and Manganese Removals by Iron Manganese Removal Processes

| City | pH | Ra-226 (pCi/L) | | | Iron (mg/l) | | | Manganese (Mg/l) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Raw | Treated | Removal | Raw | Treated | %Removal | Raw | Treated | %Removal |
| Adair | 6.7-6.9 | 6.9 | 6.7 | 3 | 0.5 | 0.01 | 80 | 0.01 | 0.01 | - |
| Eldon | 7.8 | 49 | 43 | 12 | 2.0 | 0.3 | 85 | 0.01 | 0.01 | - |
| Estherville | 7.7 | 5.7 | 5.1 | 11 | 2.0 | 0.67 | 66 | 0.24 | 0.27 | - |
| Grinnell | 7.6 | 6.7 | 5.7 | 15 | 0.7 | 0.41 | 42 | 0.01 | 0.01 | - |
| Herscher | 7.6-8.3 | 14.9 | 6.6 | 56 | 0.2 | 0 | - | 0.47 | 0.02 | 96 |
| | | 14.5 | 6.4 | 56 | 0.4 | 0 | - | 0.41 | 0.01 | 98 |
| | | 14.9 | 6.9 | 54 | 0.1 | 0 | - | 0.48 | 0.01 | 98 |
| | | 14.3 | 6.9 | 52 | 0.1 | 0 | - | 0.39 | 0 | 100 |
| | | 14.0 | 6.9 | 51 | 0.1 | 0 | - | 0.45 | 0 | 100 |
| | | 13.9 | 6.8 | 51 | 0.1 | 0 | - | 0.63 | 0 | 100 |
| | | 13.9 | 7.3 | 47 | 0.2 | 0.1 | - | 0.44 | 0.13 | 70 |
| | | 14.1 | 6.3 | 55 | 0.1 | 0 | - | 0.53 | 0.02 | 96 |
| | | 14.3 | 6.5 | 55 | 0.1 | 0 | - | 0.50 | 0 | 100 |
| Holstein | 7.4-7.6 | 13 | 7 | 46 | 1.8 | 0.09 | 95 | 0.15 | 0.01 | 93 |
| Stuart | 7.6-7.9 | 16 | 12 | 25 | 0.94 | 0.03 | 97 | 0.01 | 0.01 | - |

Source: Bennet, D.C. et al. (1976), Schliekelman, R.J. (1976), Brinck, W.L. et al. (1978)

## Table 7

### EPA Uranium Removal Field Study by Anion Exchange

| | Raw Water* Uranium (microgram/liter) | Treated Water Uranium (microgram/liter) | Bed Volumes† Treated | Capacity†† gram/liter |
|---|---|---|---|---|
| Ft. Lupton, CO | 35. | 35. | 11,867† | 0.112 |
| Brighton, CO | 23. | 23. | 24,181† | 0.144 |
| Marshdale, CO | 28. | <0.1 | 21,601 | — |
| Cove, AZ | 64. | 63. | 16,702† | 0.272 |
| Church Rock, NM | 52. | 0.1 | 10,830 | — |

*Uranium 238/235 ratio not determined. If uranium 238/234 is 1/1 then 1 microgram/liter equal 0.67 pCi/L

†Bed Volumes treated when effluent first reaches influent uranium level
1 BV = 1.88 gallons

††Grams uranium removed per liter of resin (g/L)

## Table 8

### Removal of Uranium From Pond Water by $Ca(OH)_2$ Treatment[a]

| | $Ca(OH)_2$ doses (mg/L) | | | | |
|---|---|---|---|---|---|
| | 50 | 100 | 150 | 200 | 250 |
| % U Removed | 86 | 85 | 87 | 87 | 90 |
| Final pH | 10.6 | 11.1 | 11.3 | 11.5 | 11.5 |

[a] Initial U Concentration: 83 micrograms/liter.

Source: Bondietti, et al., 1982.

## Table 9

### Suspected Uranyl Species and Charge Characteristics of Iron and Aluminum Hydroxide Flocs at Given pHs of Pond Water

| | Adjusted pH | | | |
|---|---|---|---|---|
| | 4 | 6 | 9 | 10 |
| Uranyl Species | $UO_2^{2+}$ | $UO_2CO_3^0$ | $UO_2(CO)_2^{2-}$ | $(UO_2)_3(OH)_5^0$ |
| Charges of Flocs | $(Fe,Al)(OH)_{3-x}^{x+}$ | $(Fe,Al)(PH)_3^{0}$ | $(Fe,Al)(OH)_{3+y}^{y-}$ | $(Fe,Al)(OH)_{3+z}^{z-}$ |
| Uranium Removal | Low (30) | High (88) | Low (48) | High (87) |

Source: Bondietti, et al., 1982

## Table 10

### Percent Uranium Removal by $Fe_2(So_4)_3$, $FeSO_4$, and $Al_2(SO_4)3$ Coagulants with Varying pH[a]

| Initial pH | Dosage (mg/L) | U Removed (%) | | | Final pH | | |
|---|---|---|---|---|---|---|---|
| | | $F2_2(SO_4)_3$ | $FeSO_4$ | $Al_2(SO_4)_3$ | $Fe_2(SO_4)3$ | $FeSO_4$ | $Al_2(SO_4)_3$ |
| 4 | 0.5 | 7 | 6 | 7 | 4.1 | 4.2 | 4.4 |
| | 5 | 14 | 8 | 9 | 4.3 | 4.2 | 4.2 |
| | 10 | 8 | 11 | 6 | 3.8 | 4.1 | 4.4 |
| | 15 | 13 | 21 | 15 | 4.0 | 4.2 | 4.7 |
| | 20 | 17 | 26 | 21 | 4.1 | 4.2 | 4.8 |
| | 25 | 18 | 33 | 21 | 4.0 | 4.1 | 4.8 |
| 6 | 0.5 | 16 | 14 | 7 | 6.2 | 6.1 | 6.2 |
| | 5 | 43 | 24 | 30 | 6.4 | 6.0 | 6.2 |
| | 10 | 63 | 33 | 51 | 6.2 | 6.1 | 6.1 |
| | 15 | 76 | 42 | 69 | 6.2 | 6.2 | 6.1 |
| | 20 | 84 | 52 | 80 | 6.1 | 6.2 | 6.1 |
| | 25 | 89 | 44 | 88 | 6.2 | 6.2 | 6.2 |
| 8 | 0.5 | 1 | 6 | 0 | 8.4 | 8.1 | 8.0 |
| | 5 | 4 | 7 | 2 | 8.2 | 8.1 | 7.9 |
| | 10 | 17 | 12 | 9 | 7.9 | 8.1 | 7.9 |
| | 15 | 21 | 11 | 17 | 8.0 | 8.1 | 7.9 |
| | 20 | 33 | 15 | 25 | 7.9 | 8.0 | 7.9 |
| | 25 | 43 | 20 | 48 | 7.8 | 8.0 | 7.8 |
| 10 | 0.5 | 1 | 2 | 8 | 10.0 | 10.1 | 10.0 |
| | 5 | 27 | 32 | 71 | 10.0 | 10.0 | 9.9 |
| | 10 | 83 | 57 | 95 | 9.9 | 10.0 | 9.8 |
| | 15 | 86 | 84 | 98 | 10.0 | 10.0 | 9.7 |
| | 20 | 80 | 92 | 98 | 9.5 | 9.9 | 9.7 |
| | 25 | 87 | 93 | 96 | 10.0 | 9.9 | 9.7 |

Source: Bondietti, et al., 1982

### Table 11

### EDR Test/Analytical Means

| | Feed | Brine | Product | Rejection |
|---|---|---|---|---|
| Radiometrics | pCi/L | pCi/L | pCi/L | |
| Ra 226 | 667 | 2,904 | 64 | 90.4% |
| Th 230 | 54 | 415 | 10.0 | 81.5% |
| Gross | 735 | 3,294 | 149 | 79.7% |
| Gross | 2,182 | 4,390 | 379 | 82.6% |

Source: Personal correspondence with Ionics, Inc.

Table 12

Summary of Radium Removal Technology

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| Ion Exchange | F<br>Yes | Yes | 81-97% | S,M | B<br>50-3500 pCi/L | M | Be76, Br78<br>Sc76, Ep77 | 2 | Na -<br>Corrosion -<br>Other Ions Removed + |
| Lime Softening | F<br>Yes | Yes | 80-90% | M,L | S<br>1-10 pCi/g | L | - | 8 | Na -<br>Corrosion -<br>Cont. Removal +<br>Softening + |
| R.O. | F<br>Yes | Yes | 90+% | S,M,L | L<br>2:1 3:1 | H | So80 | 3 | Corrosion -<br>TDS + |
| E.D.R. | F<br>Yes | Yes | 90% | S,M,L | L<br>3:1 | H | - | 4 | Corrosion -<br>TDS + |
| Manganese/Iron Removal | F<br>Yes | Yes | 25-50% | S,M,L | L<br>? | - | Ep77 | 4 | Iron and Manganese Removal + |
| $MNO_2$ Imp. Fiber | L<br>New | No | ?<br>90-% | S | S<br>? | - | Co68 | - | - |
| R.S.C. | L,F<br>New | No | 90+% | S,M,L | S<br>? | - | Bo 82 | - | - |
| Barium | ?<br>New | - | - | - | - | - | - | 7 | Adv. Health Effects |

Table 13

Summary of Uranium Removal Technology

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| Anion Exchange | L,P | Yes | 90+% | S,M | B | - | (Bo82, Ha82) | 3 | ? |
| Lime Softening | L,P | Yes | 80-90% | M,L | Lot of S,L | - | (Bo82) | 9 | Na -<br>Corrosion -<br>Cont. Removal +<br>Softening + |
| R.O. | L | Yes | 90+% | S,M,L | L | - | (Sa76, Bo82) | 3 | Corrosion -<br>TDS + |
| E.D.R. | No | Yes | - | S,M,L | L | - | - | 3 | Corrosion -<br>TDS + |
| Coagulation | L,F | Yes | 80% | M,L | S,L | - | (Bo82) | 8 | Complex -<br>Cont. Removal + |
| Activated Alumina | L | Yes | 90+% | S,M | L | M | (Ha82) | 7 | - |

### Table 14

### Summary of Radon Removal Technology

|  | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|
| G.A.C. | L,P,F | Yes | 90+% | S,M | S | - | (Lo82) | 2 | Radon Daughters-<br>Rad. Licensing - |
| Aeration | L,P,F | Yes | 90+% | S,M,L | G | - | (Lo82) | 2 | Radon in Air - |

## LEGEND For Tables 12, 13, 14

1 - Demonstrated Technology

F - Full Scale
P - Pilot Plant
L - Laboratory

2 - Availability

Yes
No

3 - Efficiency

Percent Removal

4 - Size

S - Small
M - Medium
L - Large

5 - Disposal of Waste

L - Liquid
S - Sludge
B - Brine
A - Solid

6 - Reliability

L - Low
M - Medium
H - High

7 - Cost - Capital, Operating and Maintenance

8 - Complexity - 1 least complex -------------- 10 most complex

9 - Benefits and Disadvantages

R.O. - Reverse Osmosis

E.D. - Electrodialysis

R.S.C. - Radium Selective Complexes

G.A.C. - Granular Activated Carbon

Table 15. Summary of Alternatives to the Disposal and Handling of Treatment Wastes From Lime Softening, Ion-Exchange, and Reverse Osmosis

| | |
|---|---|
| Discharge | -To sanitary sewer<br>-To local receiving waters<br>a) streams<br>b) oceans<br>-By wet pumping or trucking to local sanitary landfill |
| Storage | -Permanent Lagooning<br>-Sanitary Landfill<br>a) with prior temporary lagooning<br>b) with prior mechanical dewatering: vacumn filtration, centrifugation, pressure filtration, belt filter pressing, and dual-cell gravity solids concentrations<br>-Other natural or man made depressions (all with dewatering before transportation)<br>a) strip mine areas<br>b) borrow pits and quarries<br>c) others<br>-Tanks or lagoons<br>a) for settling and decanting into receiving water<br>b) for settling and pumping supernatant back to plant<br>-Evaporation lagoons<br>-Land spreading |
| Use | -Direct without drying: farmland and pasturelands<br>-With prior dewatering<br>a) farmland and pasture land<br>b) road stabilization<br>-Recovery |
| Disposal | -Direct, recharge to aquifers<br>-With prior dewatering: salt mines, coal mines, and so forth<br>-As nuclear waste<br>-In deep aquifers<br>-In oil well fields |

Source: Environmental Protection Agency, 1977, Manual of Treatment Techniques for Meeting the Interim Primary Drinking Water Regulations, EPA-600/8-77-005 pp. 65-66, Cincinnati, Ohio

Table 16

Summary of Performance and Economics of Diffused Aeration
and Granular Activated Carbon to Remove Radon from Water
for Household Use (200 gpd Demand)

| Influent Rn pCi/L | PCi/L Effluent Rn GAC | Aeration | Cost (Estimated) | | | |
|---|---|---|---|---|---|---|
| | | | Capital | | Operating | |
| | | | GAC | Aeration | GAC | Aeration |
| 15,000 | 1350-3300 | 750 | $431-757 | $890 | $19 | $60 |
| 30,000 | 2700-6600 | 1500 | $431-757 | $890 | $19 | $60 |
| 150,000 | 1200 (2) | <7500 | $1500 | $1000 | $40 | $80 |

Source: Lowry, J.D., 1983.

# FIGURE CAPTIONS

1. Figure 1 Schematic Defining Adjustments Between the Occurrence of Radionuclide in Water and User Safety and Health Requirements.
2. Figure 2 Chart Illustrating the Effectiveness of Uranium Removal by Ion Exchange (Bo82)
3. Figure 3 Chart Illustrating the Effectiveness of Uranium Removal by Ion Exchange in a Pilot Plant (Ha82)
4. Figure 4 Chart Illustrating the Effectiveness of Uranium Removal by Activated Alumina in a Pilot Plant (Ha82)
5. Figure 5 Chart Illustrating the Costs to Remove Radionuclides from Drinking Water by Various Treatment Techniques. $/1000 gallons (Ci83)
6. Figure 6 Chart Illustrating the Operating and Maintenance Costs Associated with the Removal of Radionuclides from Drinking Waters by Various Treatment Techniques. $1/year (Ci83)
7. Figure 7 Chart Illustrating the Capital Costs Associated with the Removal of Radionuclides from Drinking Waters by Various Treatment Techniques. $/year (Ci83)

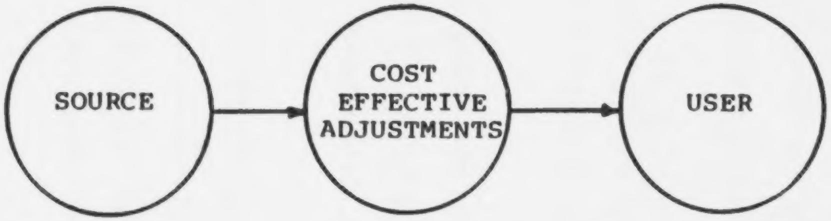
SOURCE
COST EFFECTIVE ADJUSTMENTS
USER

Figure 1. Schematic Defining Adjustments Between the Occurence of Radionuclide in Water and User Safety and Health Requirements.

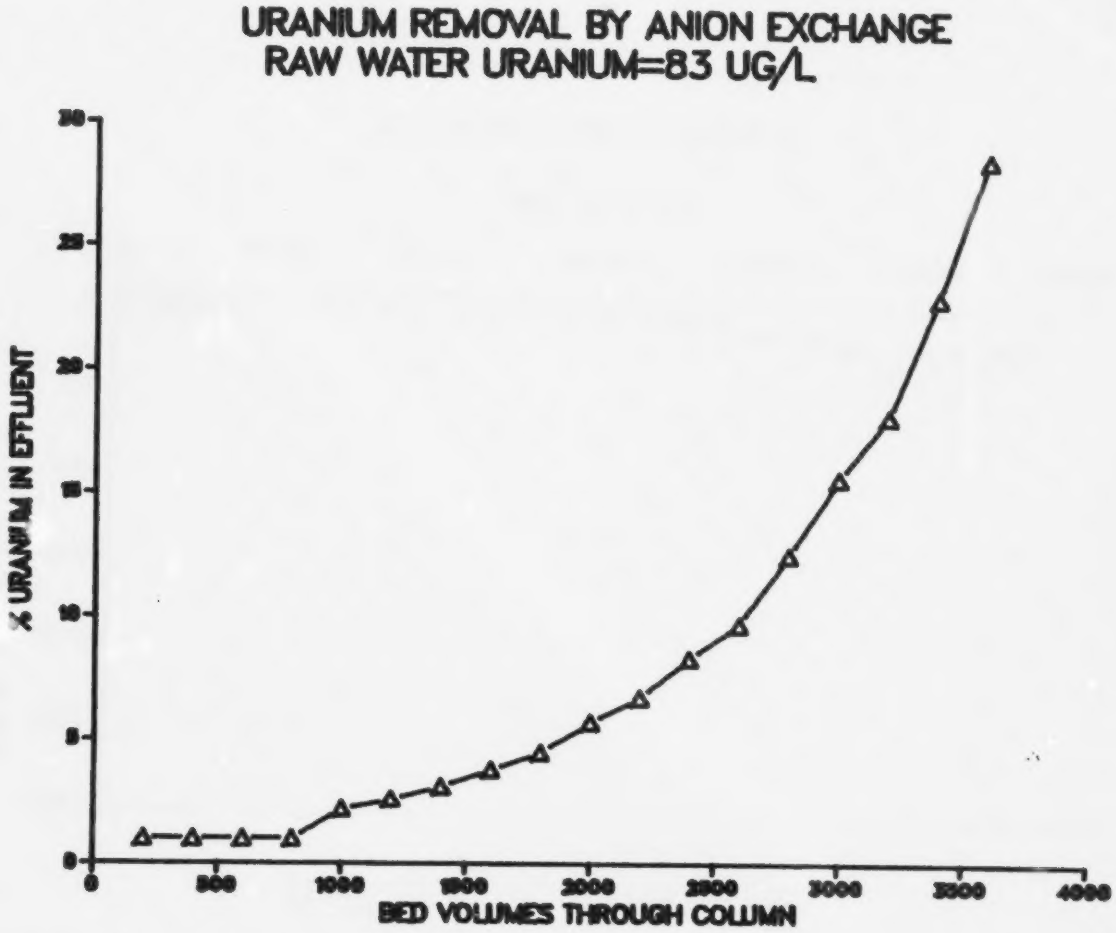

Figure 2: Chart Illustrating the Effectiveness of Uranium Removal by Ion Exchange (Bo82).

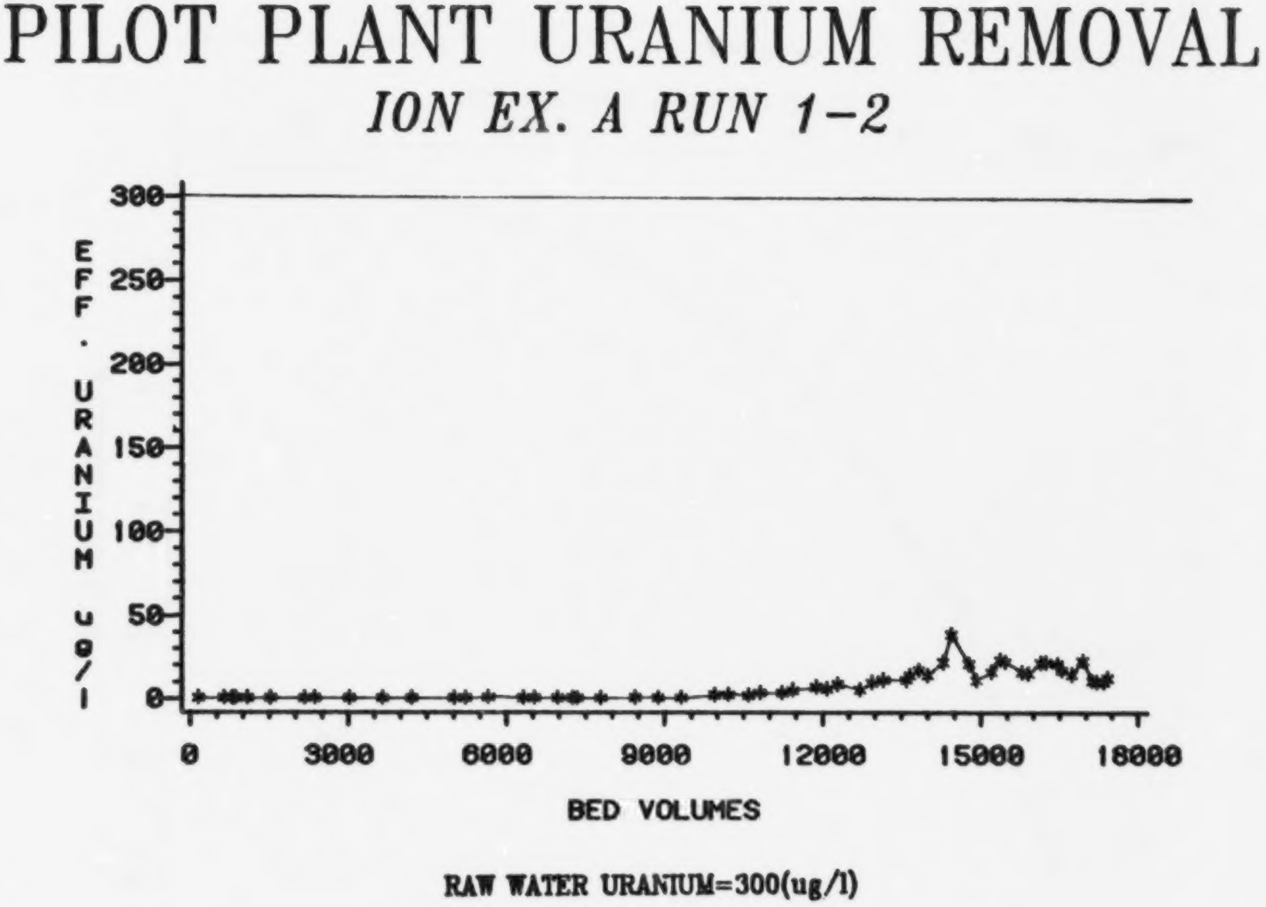

Figure 3. Chart Illustrating the Effectiveness of Uranium Removal by Ion Exchange in a Pilot Plant (Ma82).

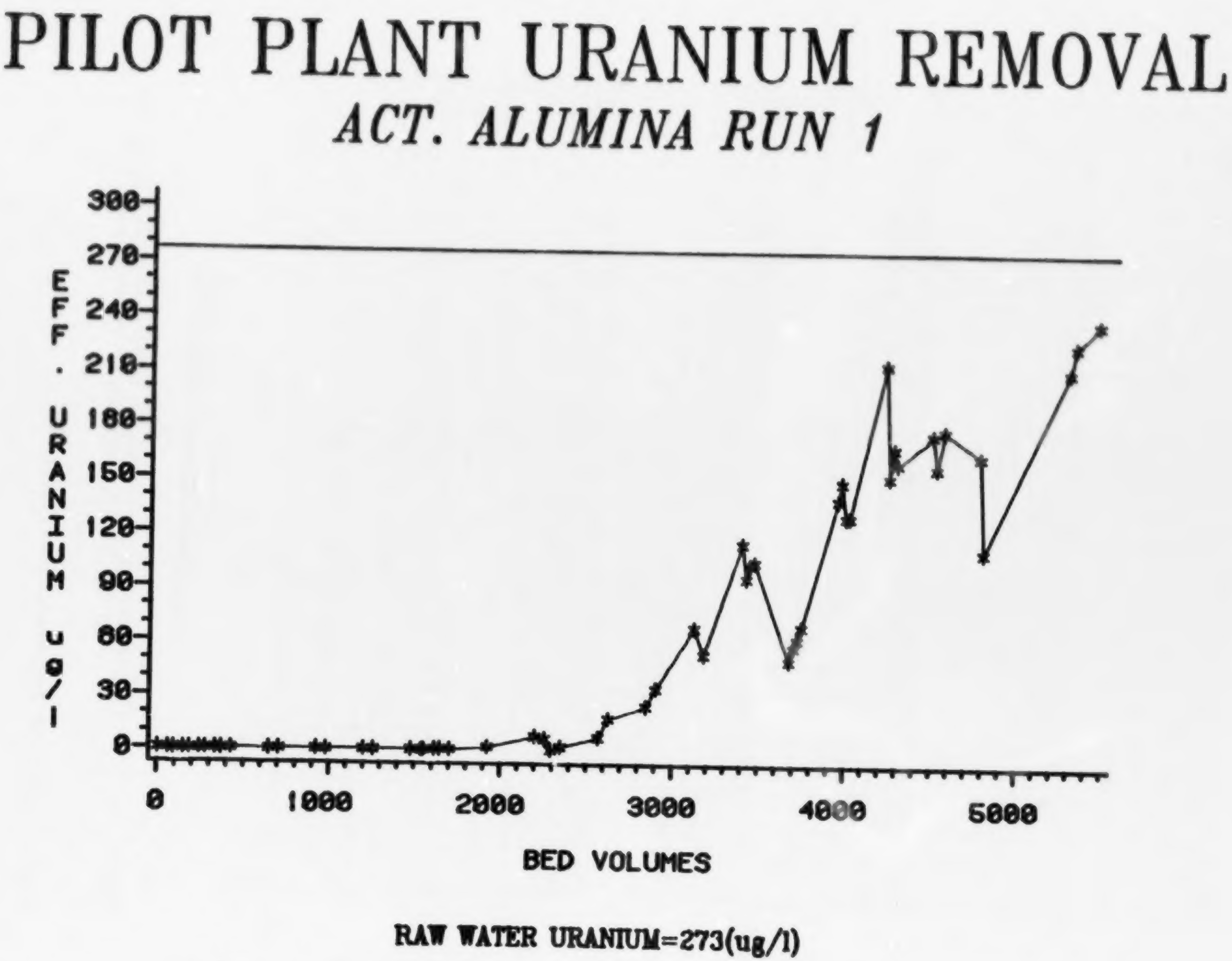

Figure 4. Chart Illustrating the effectiveness of Uranium Removal by Activated Alumina in a Pilot Plant (Ma82).

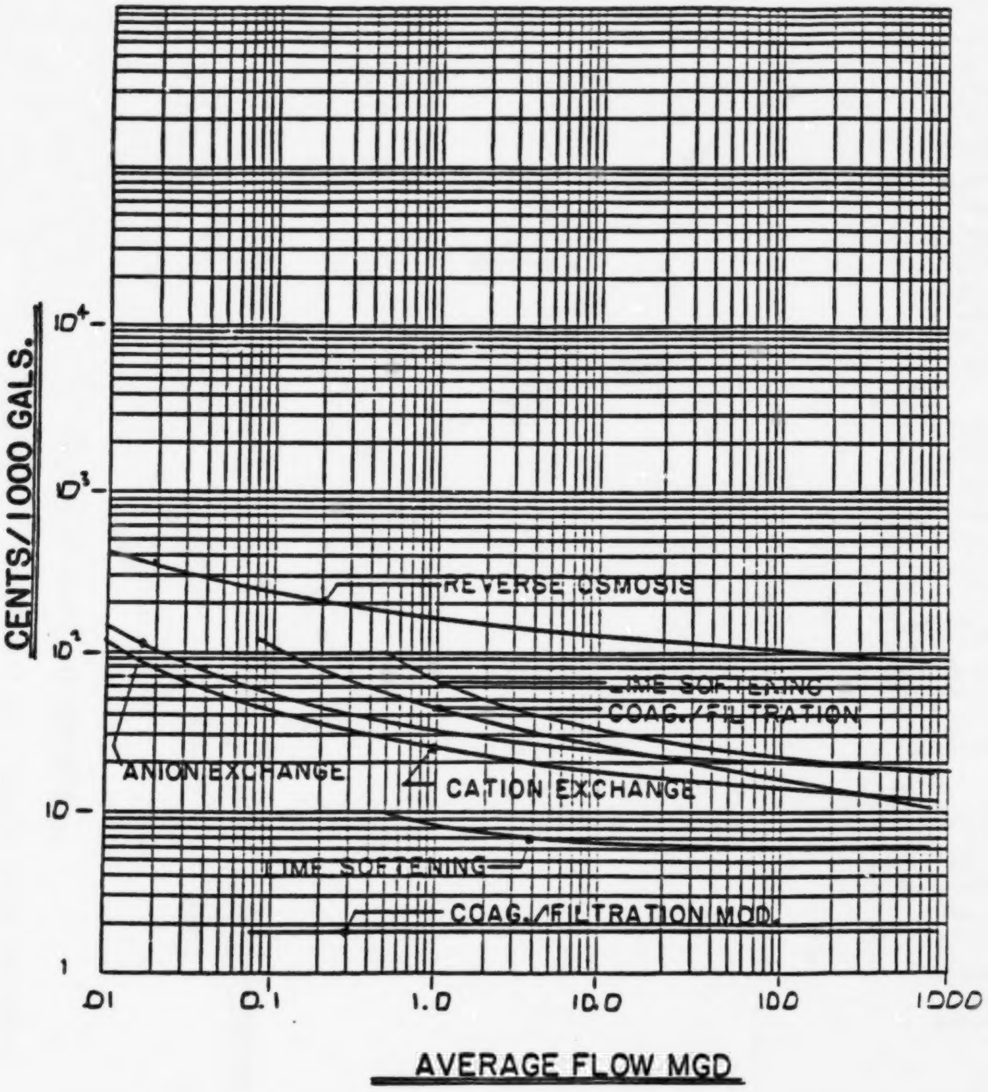

Figure 5. Chart Illustrating the Costs to Remove Radionuclides from Drinking Water by Various Treatment Techniques. $/gallons (Ci83)

Source: Ciccone and Associates, 1983.

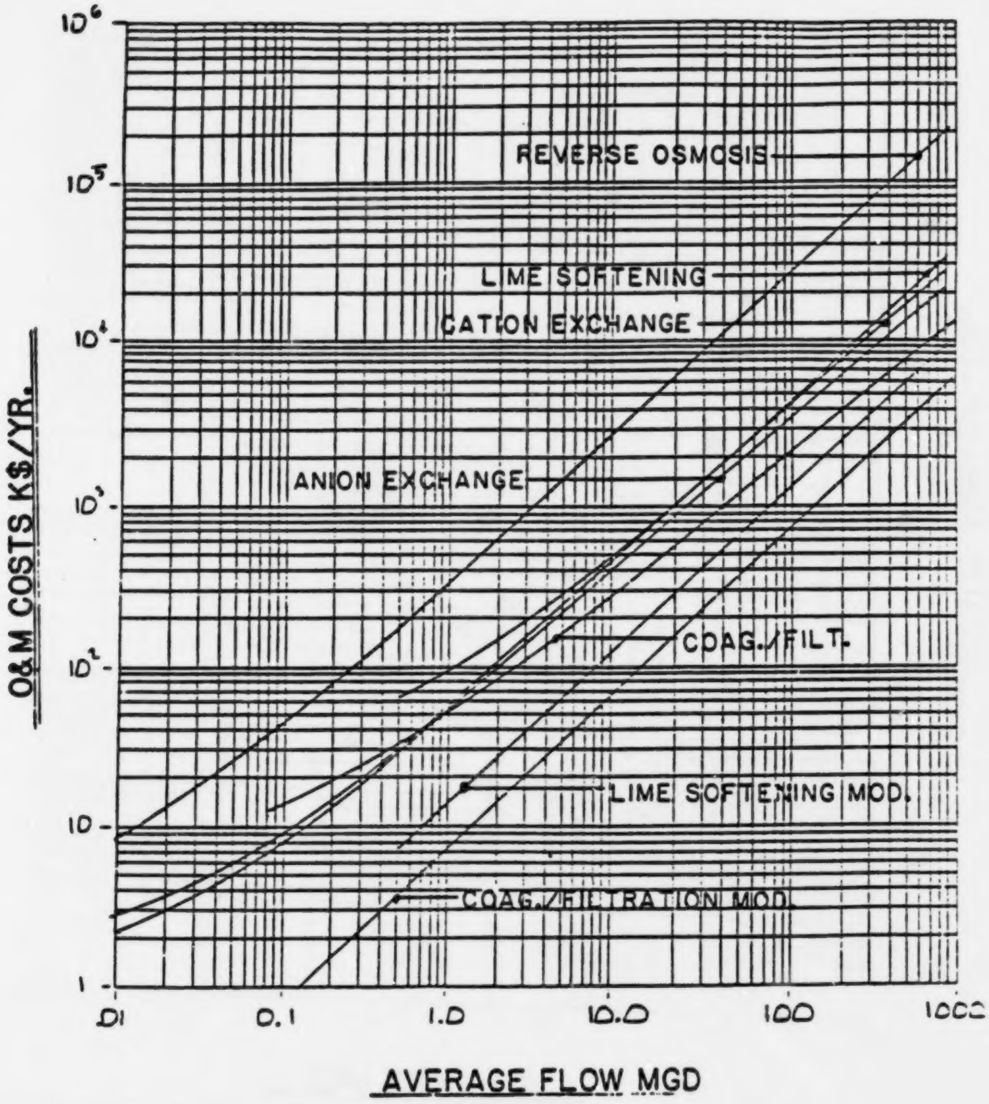

Figure 6. Chart Illustrating the Operating and Maintenance Costs Associated with the Removal of Radionuclides from Drinking Waters by Various Treatment Techniques. $/year (Ci83)

Source: Ciccone and Associates, 1983.

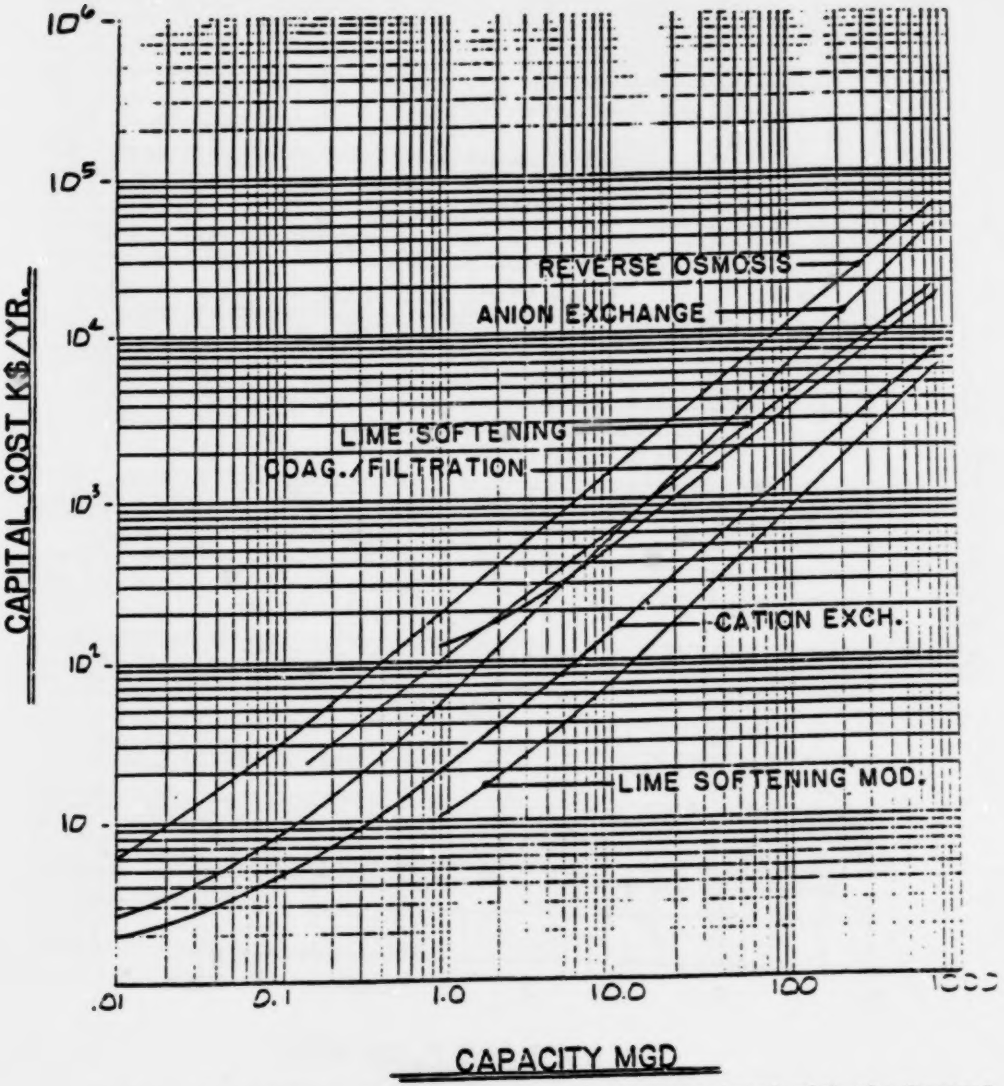

Figure 7. Chart Illustrating the Capital Costs Associated with the Removal of Radionuclides from Drinking Waters by Various Treatment Techniques. $/year. (Ci83)

Source: Ciccone and Associates, 1983.

## COMMITTEE ON HEALTH EFFECTS OF RADON

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Werner Hofmann

# HEALTH EFFECTS AND RISKS FROM RADON-222 IN DRINKING WATER

F. T. Cross
Biology and Chemistry Department
Pacific Northwest Laboratory
P.O. Box 999
Richland, WA 99352

N. H. Harley
Environmental Medicine
New York University Medical Center
500 First Avenue
New York, NY 10016

W. Hofmann
Abteilung fUr Biophysik
Universität Salzburg
Erzabt-Klotz-Strasse 11
A-5020 Salzburg, Austria

## ABSTRACT

This paper presents an evaluation of the inhalation and ingestion doses from exposure to radon and radon daughters; an overview of the human and animal health-effects data; estimations of the cancer risks from radon and radon daughter exposures; and suggested limits for radon concentrations in drinking water and indoor air. We suggest that a rounded radon-in-water concentration limit of 10,000 pCi/L can be supported by health-effects considerations alone, based on the conservative "tolerance dose" concept and other conservative assumptions regarding lung dose. A practical concentration limit (or action level) of 20,000 pCi/L has been derived by estimations of exposure distributions in the U.S. and in relation to current EPA standards for uranium-tailings-contaminated buildings. Research needed for resolution of the uncertainties in these estimates is suggested. We conclude that before a maximum contaminant level (MCL) for radon in water can be firmly established, the broader issue of setting the MCL for radon in indoor air must be addressed.

# INTRODUCTION

The inert, noble gas radon is found everywhere and sometimes occurs naturally in elevated concentrations that exceed or are substantial fractions of existing regulatory exposure standards for workers. Radon is soluble in body fluids and fats and, therefore, presents a potential hazard to the whole body. Radon in water presents a dual pathway for exposure of individuals: by ingestion, from direct water consumption, and by inhalation exposure when radon emanates from water. As developed in the following sections, the dose to the respiratory system outweighs the dose to other organ systems; therefore, derived limits on exposure to radon in drinking water are based mainly on the inhalation risk to lung.

## DOSIMETRY CALCULATIONS

Bale (1951) and Harley (1953) were the first to point out that the lung-cancer hazard from inhalation exposure to radon and radon daughters was not from the radon per se but rather from the alpha-dose delivered through lung deposition of the short-lived daughters of radon [$^{218}$Po(RaA), $^{214}$Pb(RaB), $^{214}$Bi(RaC), and $^{214}$Po(RaC')]. Two alpha-emitters, $^{218}$Po(RaA) and $^{214}$Po(RaC'), ultimately deliver the carcinogenic dose to basal cells of the bronchial epithelium, the presumed critical tissue for induction of lung cancer. The complexity of the dose estimates (required to account for daughter deposition, radioactive buildup and decay, removal by physiologic clearance processes, and physical dose calculations to specific cells in bronchial mucosa) has been detailed by many authors and considered by various national and international organizations (see Al64; Jac64, 72, 77, 80; Haq66, 67a, 67b; Pa69; Wal70, 71, 79; Har72, 81, 82; Ne74; Fr77; McP79; Jam80, 81; Ho82a, 82b; Wi82 and USPHS57, 61; FRC67; JCAE67, 69; ICRP77, 81; UN72, 77; NI71; NAS72, 80; NCRP84a).

Historically, inhalation exposure is defined in terms of the air concentration of radon daughters in working level (WL) units. A working level is defined as a concentration of short-lived radon daughters (through RaC') totaling 1.3 x $10^5$ MeV of potential alpha energy per liter of air. A working level month (WLM) is an exposure equivalent to 1 WL for 170 hours. These definitions avoid the problems of disequilibrium of the daughters and also that of whether the daughters are attached (or are unattached) to a carrier aerosol. Attached radon daughters deposit with a few percent probability to the respiratory tract surfaces, whereas unattached radon daughters deposit in the respiratory tract with nearly 100% probability. Thus, the mix of attached and unattached radon daughters is an important consideration in assessing lung dosimetry. Fortunately, the mean unattachment fraction values found in the workplace and in the environment are reasonably constant and sufficiently similar that they do not cause a large disparity in the radiological dose assessment of environmental and occupational exposures to radon daughters. The differences in other parameters that influence the radon-daughter lung dose, such as differences in daughter product equilibrium, particle-size distributions, breathing patterns, bronchial morphometry and physiological clearance processes, tend to produce somewhat compensatory doses to the basal cells of the bronchial epithelium. Thus, the pertinent radiological doses remain reasonably invariant with activity and environment and, to some extent, with age.

## Inhalation Dose

The more recent radon-daughter lung-dosimetry models, which are in substantial agreement with one another, place the bronchial epithelium exposure-to-dose conversion factor at about 0.5 rad/WLM for uranium miners. The dose per unit cumulative exposure has also been derived for environmental conditions under short-lived radon-daughter equilibrium factors(a) of about 0.7 (Har81); the finding is in close agreement for the adult male (0.71 rad/WLM), adult female (0.64 rad/WLM), the 10-year-old child (1.2 rad/WLM) and the 1-year-old infant (0.64 rad/WLM). Hofmann (Ho82a) has also found that basal-cell doses in the bronchi for infants and children are higher than for adults by about a factor of two to three in the first 10 years of life. Subsequent calculations by Harley and Pasternack (Har82) for a five-lobed human lung have yielded similar dose-factor values. The small differences in the bronchial dose for the miners and for those receiving environmental exposures primarily reflect reduced breathing rates during environmental exposures, differences in lung morphometry, differences in particle size, and the increased percentage of unattached RaA in ordinary atmospheres (~7% environmental vs ~4% in mines). These conversion factors indicate that a cumulative exposure in the nonmining environment is somewhat more effective in delivering a radiation dose to basal cells of the bronchial epithelium than are exposures under working conditions in a mine.

---

(a) The equilibrium factor is the ratio of the total potential alpha energy of the actual short-lived daughter concentrations to the total potential alpha energy that the daughters would have if they were in equilibrium with radon.

Although it seems intuitive that the reduction in breathing rate (and, therefore, radionuclide intake), which occurs under environmental exposure conditions, should reduce the dose to bronchial epithelium, the deposition onto the tracheobronchial tree increases due to the lower rate of airflow. This, combined with other compensatory differences between environmental and occupational exposure conditions, tends to result in somewhat comparable doses to basal cells for the two exposure situations.

Alternative dosimetry models (e.g.,Jac80; Jam81; Ho82a) are based on slightly different assumptions regarding particle sizes, radon-daughter unattachment fractions, breathing rates, anatomical structure, deposition equations and mucociliary transport rates. Slight changes in these physical and physiological parameters do not significantly alter the bronchial deposition and dose patterns. Differences among the models are greatest in the assumptions on basal cell depth. In the Harley-Pasternack models (Har72, 82), doses are calculated for the shallow-lying basal cells at a 22-μm constant depth below the epithelial surface, resulting in the doses being highest in the upper bronchial generations. The later calculations by Harley and Pasternack (Har82), using the Yeh-Schum morphometry (Ye80), show a more uniform distribution of dose than the earlier calculations which were based on the Weibel dichotomous model (We63). In agreement with Altshuler et al. (A164), the shallow-lying basal cells are considered by Harley and Pasternack to be the target cells in bronchogenic carcinoma. The other models utilize the basal-cell-depth distributions of Gastineau et al. (Gas72), which show decreasing epithelium thickness as the bronchial-tree branches get smaller. The variable depth assumption results in a relatively uniform basal-cell dose distribution.

In spite of the different assumptions in the models, the adult exposure-to-dose rounded, conversion factors lie within a relatively narrow range of values: 0.4 to 0.6 (Jac80), 0.3 to 1.0 (Jam81), 0.3 to 0.8 (ICRP81), 0.6 (Ho82a) and 0.5 to 0.7 rad/WLM (Har81, 82). Because conclusive evidence is lacking concerning the location of the relevant target cells, a reasonable, mean, environmental-conversion factor of 0.7 rad/WLM for bronchial epithelium has been adopted for this paper. We estimate that this value is known to within a factor of about 2.

In some models of risk for radon-daughter exposure (e.g., EPA80) there has been a tendency to artificially lower the cumulative exposure in the environment, presumably to account for the influence of decreased breathing rates on mean lung dose under nonworking conditions. In our opinion, this is neither warranted nor justifiable in view of the general agreement, at least among U.S. modelers, that the relevant doses accrue to bronchial epithelium and not to lung as a whole, and in view of the compensating factors mentioned above. Therefore, whether the inhalation exposure is environmental or occupational, the WL is given comparable weight in our subsequent treatment of risk.

The effective-dose-equivalent system of the ICRP (ICRP81) assigns equal weights to the cell dose in the bronchial and pulmonary epithelium for inhalation exposures. Although their exposure-to-dose conversion factors for bronchial epithelium (0.32 to 0.85 rad/WLM) are similar to other values for miners, the Commission's effective-dose-equivalent system has not been adopted for this paper. Instead, the critical organ/critical tissue approach of the NCRP, as reflected in their Report No. 39 (NCRP71), is utilized in our treatment of risk. For the lung, the tissue of concern is the bronchial epithelium.

## Ingestion Dose

While many investigators have addressed the dose to lung from inhaled radon and radon daughters, comparatively few have calculated the internal doses from ingested radon and radon daughters. Suomela and Kahlos (Su72) estimated the dose-equivalent based on whole-body-counting measurements of human volunteers. Using an alpha quality factor of 10, they derived doses to the stomach wall of 240 mrem/µCi $^{222}$Rn and 380 mrem/µCi $^{222}$Rn, for full and empty stomachs, respectively. These values convert to 480 and 760 mrem/µCi $^{222}$Rn, using the currently recommended value of 20 for the alpha quality factor.

Hursh et al. (Hur65) also identified the stomach wall as the tissue which receives the greatest radiation dose from ingested $^{222}$Rn. They state that the dose contribution from the total decay of the ingested short-lived daughters is negligible compared with that from the radon itself. Their calculated doses, converted to current rem units, are 412 mrem/µCi $^{222}$Rn and 440 mrem/µCi $^{222}$Rn, for full and empty stomachs, respectively.

The estimates of von Döbeln and Lindell (Vo64) include whole-body doses. Their converted values are 400 mrem/µCi $^{222}$Rn for stomach and 4 mrem/µCi $^{222}$Rn for whole body. The converted whole-body dose equivalent of Andersson and Nilsson (An64) is somewhat higher: 14 mrem/µCi $^{222}$Rn.

Current evaluations of these doses appear to be based on the earlier work (e.g., EPA77, Ka80, Sul82). The analysis of radon-ingestion models by Sullivan and Nelson (Sul82) concludes that, in spite of the many papers on radon ingestion, experimental measurements are few and somewhat contradictory. They further state that our present knowledge of the behavior of ingested radon is insufficient to support a proposed maximum contaminant level. The uncertainties identified were: (1) the unknown transit time of radon through the gastrointestinal tract wall, (2) the identification of the organs receiving the highest doses, (3) the variability in whole-body radon retention, and (4) the unaccountability for all the radon putatively ingested--up to 50% could not be accounted for in some of the experiments.

In view of the many uncertainties in the data, it is not surprising that various investigators have apparently assumed inordinately long retention times for radon in the stomach or other components of the gastrointestinal tract, in an attempt to be conservative in their estimates of the ingestion doses. Sullivan and Nelson (Su182) estimated that the ingestion doses to the gastrointestinal tract may be overestimated by as much as two-orders-of-magnitude and, therefore, they do not consider present data adequate for assessing radon-ingestion doses.

Using the previous ingestion dose estimates for illustration purposes, reasonable values range from about 400 to 800 mrem/μCi $^{222}$Rn to the stomach and from 4 to 14 mrem/μCi $^{222}$Rn to the whole body. Reasonable mean values might be 600 mrem/μCi $^{222}$Rn and 10 mrem/μCi $^{222}$Rn, respectively, to the stomach and whole body. These values can be converted to annual dose equivalents if we assume consumption of 0.5 L of untreated (including unheated and nonaerated) water per day. [The amount of untreated water consumed is considered by Suomela and Kahlos to vary between 300 and 1200 ml/day; however, many investigators prefer to use the lower number. The ICRP (1975) estimates daily tap-water consumption at 150, 100 and 200 ml for adult reference man, woman and 10-yr old child, respectively.] For a radon concentration in water of 1000 pCi/L, the calculated annual dose equivalent is about 100 mrem to stomach and 2 mrem to whole body.

If we use Kahlos and Asikainen's (Ka80) mean transfer coefficient of $10^{-4}$ for radon in water to radon in house air, 1000 pCi/L in water converts to 0.1 pCi/L in air. Using 0.7 rad/WLM as a reasonable lifetime conversion coefficient for environmental exposures, an alpha quality factor of 20, an equilibrium factor of 0.5, and continuous exposure, the 0.1 pCi/L converts to a rounded bronchial epithelium dose equivalent of 400 mrem/yr. The value of about $10^{-4}$ for the mean radon transfer coefficient has been confirmed by Hess (Hes82) for houses in Maine with 1 air change per hour. In the UNSCEAR 1977 report (UN77), the mean radon transfer coefficient was calculated to be $2 \times 10^{-4}$ for a ventilation rate of 1 $h^{-1}$. Because some houses have lower ventilation rates, it is conservative to assume that the mean radon transfer coefficient ranges from about $1 \times 10^{-4}$ to $2.5 \times 10^{-4}$. The dose-equivalent to bronchial epithelium for 1000 pCi/L in water, therefore, ranges from about 400 to 900 mrem/yr. Thus, the estimated dose to bronchial epithelium from radon in water is substantially higher than the estimated ingestion doses. The actual amount of untreated water drunk per day is, therefore, relatively unimportant, because the lung dose, multiplied by the risk factor for lung, compared to the same products for stomach and whole body, determine the concentration limits for radon in drinking water.

# RADON DAUGHTER EPIDEMIOLOGY STUDIES

## Introduction

Data from a variety of occupational and medical exposures, and from disasters, have clearly implicated ionizing radiation as a human carcinogen. While there are many epidemiology studies addressing these types of exposure, comparatively few studies have investigated the exposure to natural background radiation sources. Even those few show no significant increase in lung-cancer death rate from inhalation exposure to normally occurring levels of radon and radon daughters. Positive correlations exist for $^{226}$Ra levels in drinking water and cancer incidence (e.g., Be82), but an unequivocal association of radium and radon in drinking water and cancer incidence has yet to be demonstrated.

The most notable example of nonmining radon exposures are thorotrast patients whose lungs continually receive a constant, low-level alpha exposure from $^{220}$Rn and its daughters. A higher risk of lung tumors has not yet been demonstrated in these patients, who have received estimated doses to the large bronchi of 300 rad over a 30-year period (Va78, 83). Although these exposures are not strictly comparable to $^{222}$Rn daughter exposures, a $^{222}$Rn-daughter exposure which produces about 300 rad to the bronchial epithelium ranges between 400 and 600 WLM, depending on whether the exposures are to environmental radon or radon in the mines. These levels of exposure have an upper estimated lifetime lung cancer risk of about $10^{-1}$, using the lung cancer risk factor of $5.6 \times 10^{-3}$ for persons exposed to 1 WLM/yr for life that is developed later in this report.

The epidemiological data derived from many types of underground mining show a relatively consistent relationship between lung-cancer incidence (which is similar to the death rate from lung cancer) and exposure to radon daughters. This underlying consistency is considered to be related to the relatively narrow range of bronchial dose per WLM under varying exposure conditions.

It is difficult to assess the risk of attributable lung cancer through human epidemiological studies because the detailed information required is not always available. In the ideal case, the exposure of each miner, as a function of time, would be available; and the follow-up period would be long enough for all of the group to have died from lung cancer or other causes. From such ideal data, attributable lung cancers could be separated from those arising spontaneously or from cigarette smoking. The cumulative exposure, person-years at risk, and the number of attributable lung cancers would allow the exact calculation of a risk factor.

In reality, the data do not fulfill these requirements: estimates of exposure were often crude, and follow-up periods are not sufficiently long. Nevertheless, recognizing the limitations of the data, it is possible to estimate a mean risk factor which we can accept until improved data and further studies provide more firmly based estimates of risk.

Human data are now available from several groups of underground metal-ore miners: those in the U.S., Canadian and Czechoslovakian uranium mines, Swedish and British iron mines, Swedish lead and zinc mines, and Newfoundland fluorspar mines. Although other potential carcinogens (such as diesel smoke, traces of arsenic or nickel, and iron ore) are found in these mines, the lung-cancer response appears to be predictable, based upon radon-daughter exposure. Some studies have divided the workers into subgroups on the basis of estimated radon daughter exposure. Eighteen subgroups were selected (Ar79) as most suitable (considering both epidemiological and environmental data) for quantitative assessment of the lower exposure levels. In addition, data on these mining populations have been reviewed by other authors and organizations (see NI71; NAS72, 80; Se76; Jo73; Ax78; Sn73, 74; Ren74; Dev64; Wr77; McP79; UN77; Ev81; Ra81; NCRP84a).

## Discussion and Summary

Present data suggest that an absolute threshold exposure for lung-cancer induction is highly unlikely. [This is also in keeping with present-day views, in radiation biology and radiation protection, that radiation-induced cancer is a stochastic (nonthreshold) process.] Evans (Ev67) and Stranden (Str80) argue that the lung-cancer mortality data at the lowest reported exposures are not statistically different from expected and that at least a "practical" threshold for radon-daughter carcinogenesis may exist. Archer et al. (Ar79) conclude from their analysis of the 18 subgroups that, if a threshold exists, it is less than the range from 20 to 30 WLM. Snihs (Sn73, 74) considers that the lowest underground exposure resulting in an apparent increase in lung-cancer deaths in Swedish miners is about 15 WLM, although he states that it is impossible to draw conclusions about the exposure-response relationship below 100 WLM. Hewitt (He79) concludes from an analysis of Canadian uranium miners that if a threshold exists, it is below 60 WLM. These varied opinions seem to indicate the possibility that environmental exposure to radon daughters (or very-low-level exposures) may result in such a small lung cancer rate as to be indistinguishable from the natural, nonradiological induction rate.

The incidence of lung cancer attributable to radon-daughter exposure that has been observed in the various mining subgroups ranges from about 1.5 to 50 cases per WLM/year/$10^6$ persons, with a present-day rounded average value of 10 x $10^{-6}$ per person per year per WLM, for estimated mean follow-up times ranging from about 14 to 48 years. This average value has been accepted in the lung-cancer prediction model of Harley and Pasternack (Har81) as reasonably realistic when their modeled data are also compared to background (normally occurring) lung-cancer incidence in nonsmokers from environmental exposure to radon. The NCRP (NCRP84a) has calculated the mean and standard error of the estimated lung-cancer incidence to date to be 12 ± 2 per WLM/year/$10^6$ persons from a data base of 23 exposure groups. The 95% confidence interval of the mean, therefore, ranges from 8 to 16 per WLM/year/$10^6$ persons.

In estimating the effect of radon-daughter exposure at environmental levels (normally, less than about 20 cumulative WLM per lifetime), the attributable risk at high exposures, derived from the mining data, must somehow be extrapolated to the low-exposure region. In keeping with prudent, conventional practice, the extrapolation is linear, even though some studies suggest that exposures may be even more efficient in inducing lung cancer as the exposure rate approaches background levels (Ar78; NAS80). This hypothesis is in contrast to the possibility mentioned above that very-low-level exposure to radon daughters does not result in distinguishable lung cancer. Recent track structure analyses suggest that lung cancer incidence is a quadratic or cubic function of dose when allowing for concomitant cell killing (Ho83). If so, risk at low doses is very much smaller than indicated by the linear extrapolation.

While risk may be extrapolated to low exposure levels, the accuracy of any risk estimate depends not only on the extrapolation model but also on the accuracy of the measurement, or estimation of exposure. From a statistical viewpoint, therefore, risk estimates for low level exposures would have a large confidence interval and, thus, relatively poor accuracy.

## Influence of Modifying Agents

The effect of cigarette smoke in modifying radiation-induced cancer probabilities remains uncertain at this time. During periods of relatively short follow-up (15-25 years), cigarette smoking has been associated with a markedly increased incidence of lung cancer in miners. During 30- to 60-yr periods of follow-up after initial exposure, lung-cancer incidence is reported to be either somewhat greater among nonsmokers than smokers (Ax80) or about the same (Ra81). This latter evidence is in agreement with the results of studies of beagle dogs that had comparable radon daughter exposures and were exposed to cigarette smoke (Cr78): dogs that "smoked" had fewer respiratory-tract tumors than dogs that did not "smoke." The current evidence suggests that the principal role of cigarette smoking in uranium miners is to accelerate the appearance of lung cancer induced by radiation, although even this idea has recently been challenged (Sa82). However, the issue cannot be considered resolved as yet, and the role of smoking at low occupational or environmental radon-daughter levels is unknown.

Circumstantial evidence appears to rule out ore dust and diesel exhaust as important contributors to the observed incidence of lung cancer among underground uranium miners (FRC67). This evidence is also supported by data from the animal experiments discussed in the following section.

Finally, the co-influence, if any, of other physical, chemical or biological agents at low occupational or environmental radon-daughter levels is unknown.

# ANIMAL STUDIES

## Introduction

Animal studies have been conducted for several decades to identify the nature and levels of uranium mine-air contaminants that were responsible for producing the lung cancers observed among uranium-mining populations. Many of the initial studies were concerned with early effects or short-term pathological changes (Jan26; Re39; Ja40). Exposures were based primarily on radon gas concentrations, giving little or no information on the radon-daughter concentrations, which subsequently were shown to contribute the greatest radiation dose to the lung. The early studies (Hu39; Raj42a, 42b; Ku59), in which lung tumors were produced, were methodologically or statistically inadequate to show an unequivocal association of lung tumors with exposure to radon and/or radon daughters.

Beginning in the 1950s, a growing concern emerged that the increased incidence of respiratory cancer observed in the European uranium-mining population would also be found in the U.S. mining population (SS55; Wa64). Systematic studies were subsequently begun in the U.S. to identify the agents responsible for the increased incidence of lung cancer in miners and to develop exposure-response relationships in animals. Investigators at the University of Rochester began to focus attention on the biological and physical behavior of radon daughters as well as their contribution to the radiation dose to the respiratory tract (Ba51; Harr54; Mo55). Shapiro (Sh54) exposed rats and dogs to several levels of radon alone and in the presence of radon daughters attached to room-dust aerosols. He also showed that the degree of attachment of radon daughters to carrier dust particles was a primary factor in influencing the α-radiation dose to the airway epithelium. He demonstrated that this dose was due primarily (>95%) to the short-lived radon daughters RaA ($^{218}$Po) and RaC' ($^{214}$Po), rather than to the parent radon.

In 1953, Cohn et al. (Co53) reported the relative levels of radioactivity found in the nasal passages, in the trachea and major bronchi, and in the other portions of rat lungs after exposure to radon and/or radon daughters. The respiratory tracts of animals that inhaled radon plus its daughters contained 125 times more activity than those of animals that inhaled radon alone.

Beginning in the mid 1950s, Morken initiated a pioneering series of experiments (Mo66, 73a, 73b) to evaluate the biological effects of inhaled radon and radon daughters in mice; later experiments used rats as well as beagle dogs. The essentially negative biological results of these studies (due primarily, we believe, to inadequate follow-up times in the experiments) suggested that α-irradiation is inefficient in producing tumors in the respiratory system. The only apparently permanent late changes occurred in the alveolar and respiratory bronchiolar regions of the lung for a wide range of exposure levels and for observation times to 3 years in the dog and 1 and 2 years, respectively in the rat and mouse. Furthermore, injury in the bronchial tissue was quickly repaired after irradiation ceased. The carrier aerosols used in these experiments were more typical of environmental aerosols than of those found in the mines.

In the late 1960s and early 1970s, other studies in France and the U.S. were initiated, which later proved successful in producing lung tumors from inhaled radon daughters. The French exposed rats either to radon daughters alone or in combination with stable cerium, uranium-ore dust or cigarette smoke to produce tumors in the lung (Pe70; Ch74, 80). The later U.S. studies were designed to systematically determine the pathogenic role of radon daughters alone, or in various combinations with uranium-ore dust, diesel-engine exhaust and cigarette smoke. These studies involved lifespan exposures of beagle dogs and Syrian Golden hamsters and chronic exposures of rats (Cr78, 82).

A discussion of the biological effects in animals of inhaled radon and radon daughters is included in the recent ICRP Publication 31 (ICRP80). In general, the lung cancers in rats were noted to be about half bronchogenic and half bronchioloalveolar in origin, in contrast to the nearly exclusive bronchogenic origin of human lung cancers. Extrapulmonary lesions were not a significant finding in the radon inhalation studies. An even more detailed presentation of the animal studies is presented in NCRP Report No. 78 (NCRP84a).

## Discussion and Summary

The animal studies have provided considerable data confirming the human epidemiology studies:

(1) In rats, primarily, tumor production per WLM at very high exposures was lower than at moderate exposures (Cr82; Ch80). The lowest attributable lung cancer rates per unit exposure were observed in miners exposed to the highest radon-daughter levels in underground mines.

(2) In both the human and animal studies, tumor production appeared to increase with decrease from high exposure rate (Ch81; Cr82); the influence of exposure rate is unknown at current occupational and environmental exposure levels.

(3) In a small group of Swedish zinc/lead miners, a lower lifetime incidence of lung cancer was observed in those who smoked and were exposed to radon daughters than in the nonsmokers. This is tentatively ascribed to the protective effect of increased mucus production from smoking (Ax78) or of the thickened mucosa resulting from smoker's bronchitis. A similar result was observed in dogs (Cr78). In rats, tobacco smoke was found to be cocarcinogenic with radon daughters when exposure to the smoke followed completion of exposure to the daughters (Ch80). This effect was not observed, however, when smoking preceded the radon-daughter exposure (Ch81). Such disparities may partially explain discrepancies in interpreting epidemiological data.

(4) Emphysema and fibrosis have been attributed to radon-daughter exposure in animals-- hamsters, rats and dogs (Stu78; Cr78)--and underground miners. Simultaneous exposure to ore dust or diesel exhaust increased the incidence of these lesions but did not appear to increase the number of tumors produced by exposure to radon daughters (Cr78, 82; Ch81).

(5) For equivalent, cumulative, radon-daughter exposure, the older the animal at the start of exposure, the shorter the latent period (Ch81). In humans, the highest risk coefficient calculated, about 50 x $10^{-6}$

lung cancers per year per WLM, is that for persons first exposed when over 40 years of age (NAS80).

(6) The predictions of the various dosimetric models appear to be borne out in the various species. The tumors induced in experiments with animals are commonly more distal than those in humans. Desrosiers' (De78) modeling of Syrian Golden hamster lungs showed that peripheral basal and Clara cells may receive doses greater than, or approximately equal to, those received by basal cells in the central airways. Human tumors have appeared almost exclusively in the upper generations of the bronchial tree. Some absorbed-dose calculations show that basal cells in human upper airways, at about the level of the segmental bronchi, receive the highest dose from radon daughters (e.g., A164; Har72, 81).

(7) Lifetime risk coefficients are similar in both animals and humans. The coefficients based on rat data appear to range between 1 and $4 \times 10^{-4}$ per WLM for all tumors (benign and malignant) at cumulative exposures less than 5000 WLM (Ch81, Cross, unpublished data). At exposures considerably lower than where lifespan is significantly shortened (<500 WLM), the lifetime risk coefficient appears to be about $2 \times 10^{-4}$ per WLM for malignancies and ranges between 2 and $4 \times 10^{-4}$ for all tumors. Data are as yet insufficient to determine a value for exposures below 100 WLM.

## LUNG-CANCER RISK-PREDICTION MODEL

The predictive model of Harley and Pasternack (Har81, RPC80) has been adopted for lung-cancer risk predictions because it allows risk coefficients to be developed for various age groups and exposure periods. This absolute-risk model is also used by the NCRP in their report on the evaluation of occupational and environmental exposures to radon and radon daughters in the United States (NCRP84a). It is based upon the most recent estimates of lung-cancer deaths among underground miners and accounts for the apparent increase in lifetime risk with increasing age at first exposure and duration of exposure (an effect not possible with a relative-risk model), as noted in the epidemiological studies of these miners. Although the model represents a reasonable uranium-miner lung-cancer response, the validity of extrapolation to environmental levels is unknown.

The adopted average yearly risk coefficient obtained for all exposure categories and all age groups ($10 \times 10^{-6}$ lung cancers per year per WLM) corresponds to a lifetime risk (to age 85 years) of about 1 to $2 \times 10^{-4}$ per WLM, dependent, of course, on activity, age at first exposure and duration of exposure. For comparison, the ICRP (ICRP81) has adopted a range for lifetime risk of 1.5 to $4.5 \times 10^{-4}$ per WLM, based primarily on Czechoslovakian underground-mining data. Evans et al. (Ev81) estimated the lifetime risk (which, they state, is applicable to the general population) to be 1 x $10^{-4}$ per WLM from U.S. and Czechoslovakian uranium-miner epidemiological data. Twice this value (2 x $10^{-4}$ per WLM) was adopted by Jacobi (Jac77) as the lifetime risk applicable to all types of miners; it is used by Cliff et al. (Cl79) to model lung-cancer incidence from environmental exposure. UNSCEAR (UN77) has reviewed the data in uranium miners in Canada, the U.S. and Czechoslovakia, in Swedish nonuranium miners and in iron miners in the United Kingdom. UNSCEAR indicates that the probable lifetime lung-cancer risk is 2 to 4.5 x $10^{-4}$ per WLM. The BEIR III report contains lung-cancer data in U.S., Canadian and Czechoslovakian uranium miners, Newfoundland fluorspar miners and Swedish metal miners (NAS80). The range of risk for all groups was expressed as 6 to 47 x $10^{-6}$ lung cancers per person per year per WLM, the upper value being for the Czech miners who began exposures at age 40 or older. If we assume that lung-cancer expression takes place over a 30-year interval (to account for the BEIR report's exclusion of the latent period in developing the yearly rate of risk), the 6 to 47 x $10^{-6}$ per person per year per WLM reduces to a range of lifetime risk from about 2 to 14 x $10^{-4}$ per WLM. The high value is considered, by BEIR, to be the most likely risk estimate, at exposure concentrations of about 1 WL, for those over 65 years of age at lung-cancer diagnosis. This value is higher than any of the other reported values and cannot be reconciled until a closer examination of these miners has been made regarding age at first exposure, cumulative exposure, exposure rate, smoking history, etc.

Except for the high value of BEIR, the lifetime risk estimates for lung cancer attributable to radon-daughter exposure (per WLM) appear to be reasonably consistent, considering the difficulty in estimating this quantity without complete follow-up, and the various methodological problems encountered in epidemiological studies.

Other features of this predictive model are that lung cancer does not occur either before a 5-year latent interval or before age 40, and that risk is corrected from year of exposure by an empirical exponential, cellular repair factor (20-year half-time). An appropriate life-table value is utilized to account for competing risks of death.

Although the basic incidence data from the underground-mining epidemiological studies cannot be applied directly to environmental situations (because patterns of exposure differ), a common factor exists in the risk per rad for bronchial basal-cell dose. The lifetime lung-cancer risk attributable to a dose of 1 rad per year has been calculated, using the conversion factor of 0.5 rad/WLM estimated for miners (RPC80). For environmental exposure starting at 1 yr of age, the lifetime risk (for exposure to age 85 years) is calculated to be $1.3 \times 10^{-2}$. Because exposure of a population involves persons of various ages, it is sometimes necessary to know the lifetime risk of radon-daughter-induced lung cancer for a population with age characteristics typical of the United States. This value, using the 1975 age distribution for the U.S. (WHO78), is calculated to be $8.0 \times 10^{-3}$ per rad per year exposure. These risk coefficients may be used for estimating lung-cancer risk from any source of radon-daughter exposure.

Risk from a bronchial dose in rad per year to basal cells is not the most useful way to evaluate environmental exposures. Two, more useful, lifetime risk coefficients can be derived that relate risk to environmental exposure in units of WLM per year, and to an annual exposure to a radon concentration of 1 pCi $^{222}$Rn/L. These coefficients use the previously indicated, average environmental exposure-to-dose conversion factors for the adult male, female, 10-year-old child, and infant, as well as assumptions on the radon-daughter equilibrium factor. The derivation of these risk coefficients for environmental exposures is simplified considerably and contains very little error, if we accept the environmental exposure-to-dose conversion factor of 0.7 rad/WLM (which applies to adult males) for all people. The lifetime risk estimate, which includes the effect of the higher dose-conversion factor in childhood, is within 10% of this value (see Tables 2 and 3 in Har81). This conversion to WLM units places at $9.1 \times 10^{-3}$ the lifetime risk coefficient for beginning exposure at infancy; for populations with ages characteristic of U.S. inhabitants in 1975, it is $5.6 \times 10^{-3}$ per WLM per year for lifetime risk and lifetime environmental exposure.

For the case of exposure measured as radon concentration over time, the average annual bronchial dose to an adult male from radon daughters associated with exposure to 1 pCi $^{222}$Rn/L (assuming he is active 16 hours per day and rests 8 hours per day) is 0.27 rad/year (RPC80). Thus, the lifetime risk for annual exposures to 1 pCi/L is calculated to be $3.6 \times 10^{-3}$ for exposure beginning at infancy and (under the same exposure conditions) $2.1 \times 10^{-3}$ for populations of mixed age. Table 1 summarizes the lifetime lung-cancer risk-coefficient data.

A uniform risk/year lung-cancer-prediction model has also been developed by Harley and Pasternack (Har81) but was rejected as being unrealistic because lifetime lung-tumor risk was found to decrease markedly with increasing age at first exposure. The ratio of the two lung-cancer prediction models (uniform risk/year model ÷ decreasing-risk model) decreases with both duration of exposure and age at first exposure. It is estimated from the data in Table 1 of Harley and Pasternack (Har81) that the estimate of lifetime risk from radon-daughter exposures would be increased by approximately two had the uniform risk/year model been employed instead of the decreasing risk model.

The environmental lifetime risk coefficients in Table 1 are based on an unattached RaA/Rn ratio of 0.07 and an equilibrium factor of about 0.7. The risk coefficients can be adjusted for other unattachment fractions and radon-daughter disequilibrium conditions. The radon-daugher equilibrium factor is considered to be the more important adjustment for environmental exposures. Under some conditions of exposure, the equilibrium factor is very low; the use of the radon-gas risk coefficients would then produce unnecessary conservatism in the estimated lung-cancer predictions. On the other hand, the use of the above radon-daughter risk coefficients when equilibrium factors are low, results in underestimation of the lung-cancer risk. Table 2 provides data for adjusting the risk coefficients for other radon-daughter disequilibrium conditions.

## Lifetime Risk From Environmental Exposure

Lifetime lung-cancer risk to populations from continuous environmental radon and radon-daughter exposure may be calculated using any of the four coefficients described above, depending on the units of exposure. For example, a lifetime risk for lifetime exposure to the average, outdoor $^{222}$Rn concentration of 0.2 pCi/L (St80; Ge80) is $7 \times 10^{-4}$, 0.07%. For comparison, Evans et al. (Ev81) calculate a value 1/3 lower for equivalent concentrations. Indoor concentrations of radon are almost always higher than those outdoors because vertical mixing cannot take place in the former case. Furthermore, indoor radon levels may be enhanced, in some cases, by elevated $^{226}$Ra concentrations in building materials and radon in the water and fuel supplies. The references cited above indicate that the average value for indoor radon concentrations ranges between 0.6 and 0.8 pCi/L, excluding basement concentrations. Using these data, a typical average environmental exposure for single-family dwellings approximates 0.5 pCi/L (accounting for both indoor and outdoor exposures), which would result in a lifetime lung-cancer risk of $2 \times 10^{-3}$, 0.2%. Because a significant fraction of the U.S. population resides in multistory buildings (Har81), and indoor levels in the U.S. relate primarily to proximity to ground beneath the structure, the true average environmental risk might be expected to lie between these two values (e.g., about 0.13%).

Published U.S. annual death rates for lung cancer among nonsmokers have ranged from about $23 \times 10^{-6}$ (Ca76) to $47 \times 10^{-6}$ (Hae58), on average, for males and females. These values can be multiplied by 45 to calculate the lifetime risk (ages 40-85), which yields 0.1 to 0.2%. The recent estimates by Enstrom and Godley (En80) and by Garfinkel (Ga80) give an average, rounded value for the lifetime risk of 0.6%.

It is impossible to confirm the accuracy of the lung-cancer-risk prediction model for environmental background exposures to radon daughters. The model does project, however, that approximately 20 to 100% of the background (nonsmoking) lung-cancer incidence can be attributed to environmental radon-daughter exposure. The value is probably closer to 20%, because of the generally accepted uncertainty in the earlier data on nonsmoker lung-cancer rates.

## Accuracy of Risk Prediction from Model

Myers and Stewart (My79) have speculated on an underlying lung cancer incidence in uranium miners that is not exposure-dependent and may represent the effect of other carcinogens. It is this factor, among others, that caused Evans et al. (Ev81) to conclude that $10^{-4}$ per WLM was the upper boundary for the lifetime risk for nonminer exposures. They state that any value greater than this would be incompatible with both British and U.S. epidemiological evidence. Exposure to environmental cocarcinogens may similarly confound the interpretation of population exposures. Therefore, the risk factors may be similar for all radon and radon-daughter exposures.

The prediction model utilizes a lifetime risk coefficient somewhat higher than the Evans et al. value and concludes that a significant percentage (20 to 100%) of the lung-cancer incidence in nonsmokers may be due to background radon exposures. The prediction model is, therefore, not unreasonable from the standpoint of grossly overestimating the background incidence. On the other hand, the upper range of the lifetime risk coefficients of ICRP and UNSCEAR, $4.5 \times 10^{-4}$/WLM, would indicate that the true values may be underestimated by as much as a factor of three. An increase in lifetime risk, however, would also proportionally increase the predicted incidence from background radon exposures.

Another factor to consider is that a large percentage of the miners studied are still alive; therefore, one might expect that the lifetime risk per WLM would increase over the values used in this paper. This possibility has been considered by the NCRP (NCRP84a), which concludes that, at the present declining rate of appearance of lung cancer, the total risk, at least for the U.S. mining group, would not double. All factors considered, therefore, we cannot conclude from the available evidence what the accuracy of the prediction model is at this time. Because we assume, in our subsequent treatment of risk, continuous exposure from infancy to the radon derived from water, along with other conservative assumptions, we believe that the prediction model provides reasonable estimates of lifetime risk for deriving concentration limits for radon in water.

ICRP organ-specific risk factors have also been employed in subsequent sections for a comparison with the lung-cancer-prediction model; the lifetime risks predicted by the ICRP are approximately 60% higher than the values derived from the lung-cancer prediction model. Insofar as the lung dosimetry and ICRP risk factor for lung represent reality, it would appear that the two methodologies for lung-cancer estimation are reasonably comparable.

## CALCULATION OF CANCER RISKS FROM EXPOSURE TO RADON IN DRINKING WATER

### Lung-Cancer Risk

Indoor radon and radon-daughter concentrations, derived from the water supply, are multiplied by the appropriate lifetime lung-cancer risk coefficients for determining the attributable risk of lung cancer from radon and radon-daughter exposures. For example, the lifetime risk coefficients of $3.6 \times 10^{-3}$ per pCi/L radon concentration and $9.1 \times 10^{-3}$ per WLM/yr (considered appropriate for exposures beginning at infancy) would be multiplied by the mean radon concentration or the mean radon-daughter exposure rate and the number of persons exposed, to provide an estimate of the total number of lung cancers produced. The risk coefficients applying to exposure beginning at infancy are employed rather than those applying to populations of mixed ages.

Regarding actual indoor exposures, Bogue (Bo59) has stated that during the course of a single year, 20-22% of the inhabitants of the United States move from one house or apartment to another. Not more than 2% of the adult population will spend an entire lifetime in the same dwelling, and less than 15% will spend a lifetime in the same county. The tendency to relocate is often least frequent among children and the elderly, and most frequent among those between 17 and 32 years of age. Mobile homes and apartment units have high rates of turnover, whereas middle- and upper-class conventional-type homes have the lowest. The average occupancy time is probably about 5 years for all ages of residents and all types of dwellings. Graphically expressed, distribution of residency time versus frequency would probably appear lognormal.

The significance of population mobility is that the additional individual lung-cancer risk for residents in houses with high radon concentrations is small if the duration of residency is short. Population mobility tends to distribute the cancer risk among a greater number of persons--those who may at some time reside in one of the dwellings with increased levels of indoor radon.

In view of the risk-coefficient adjustment factors of Table 2 and the fact that indoor equilbrium factors for radon daughters can be considerably less than about 0.7 (Ev69), we prefer to use the rounded, lifetime lung-cancer risk coefficients of $3 \times 10^{-3}$ per pCi/L radon concentration and $1 \times 10^{-2}$ per WLM/yr exposure rate, for lifetime exposure. Thus, using the range of mean transfer coefficients of 1 to $2.5 \times 10^{-4}$ for radon in water to radon in house air, the lifetime lung-cancer risk factor for continuous indoor exposure converts to 3 to $7.5 \times 10^{-7}$ per pCi/L radon concentration in water.

For comparison, the ICRP (ICRP77) has chosen $2 \times 10^{-5}$/rem as an occupational risk factor for lung cancer based primarily on external radiation exposures. Based on the previously derived lung dose-equivalent of 0.4 to 0.9 mrem/ year/pCi/L radon concentration in water, and a 60-year dose-accumulating interval, the projected lifetime lung-cancer risk factor for continuous indoor exposure ranges from 5 to $11 \times 10^{-7}$ per pCi/L radon concentration in water.

Stomach and Whole-Body Cancer Risks

According to the ICRP (ICRP77), there is evidence that radiation is carcinogenic to the stomach at moderate doses. Although there is as yet no value for the stomach risk factor, the ICRP considers that it is likely to be low. They further estimate that no single "other" tissue (which includes the stomach) has a risk coefficient exceeding $10^{-5}$/rem. Based on the previously calculated annual dose-equivalent to the stomach of about 100 mrem per 1000 pCi/L radon concentration in water, and an assumed 60-year dose-accumulating interval, the lifetime accumulated dose-equivalent to stomach is 6 rem per 1000 pCi/L of radon in water, or $6 \times 10^{-3}$ rem per pCi/L. The lifetime risk to stomach, therefore, is estimated not to exceed $6 \times 10^{-8}$ per pCi/L radon concentration in water, especially in view of the previous discussion regarding the possibility that ingestion doses are grossly overestimated.

The ICRP also estimates that the mortality risk coefficient for all radiation-induced cancers from uniform, whole-body irradiation is about $10^{-4}$/rem. Based on the previously calculated annual dose equivalent to whole body of about 2 mrem per 1000 pCi/L radon concentration in water, and an assumed 60-year dose-accumulating interval, the lifetime accumulated dose-equivalent to whole body is 0.12 rem per 1000 pCi/L of radon in water, or $1.2 \times 10^{-4}$ rem per pCi/L. The rounded lifetime risk to whole-body, therefore, is estimated to be $1 \times 10^{-8}$ per pCi/L radon concentration in water.

## Summary of Calculated Cancer Risks from Radon in Drinking Water

Table 3 presents a summary of the estimated lifetime and annual cancer-death risks associated with drinking water containing 1 pCi/L radon concentration.

The total lifetime cancer-death risk is on the order of 4 to 8 x $10^{-7}$ per pCi/L radon in drinking water; the annual risk is on the order of 8 to 18 x $10^{-9}$ per pCi/L of radon in drinking water. These estimated risk coefficients are considered conservative, as they assume continuous exposure from infancy to the radon in, and from water. In fact, it is unusual for a person to occupy a structure 100% of the time, or always during the time of water usage.

For comparison, the ICRP (ICRP77) has chosen an acceptable level of risk for stochastic phenomena in the range of $10^{-6}$ to $10^{-5}$ per year to any individual member of the public. These risks (based primarily on external radiation exposure data) imply an acceptable concentration of radon in drinking water ranging from about 60 to 1300 pCi/L. It is readily apparent that these concentrations are impractical (and therefore will not be discussed further in this paper) as very many water supplies exceed these levels (Du76; UN77, 82; Coh79). The conclusions reached by Duncan et al. (Du76), based on limited data in the U.S. and Great Britain, are that radon concentrations in water range from 0 to 30,000 pCi/L, with 25% of the locations exceeding 2000 pCi/L and 5% exceeding 10,000 pCi/L. In granitic areas, the concentrations may be an order of magnitude higher. The radon concentrations reported in the later United Nations report (UN82) range from practically zero to values up to about 2,700,000 pCi/L in some waters.

Table 4 presents total lifetime cancer risks associated with various radon concentrations in water.

## LIMITS ON EXPOSURE

It appears unlikely that we can develop reasonable limits on exposure to natural levels of radon and radon daughters by rigidly following the suggested limits on population dose or the suggested limits on population risk proposed by the various regulatory and standards-setting agencies for manmade sources of radiation. Furthermore, the variations in the concentrations of natural radioactivity, and the extensive areas of elevated levels of natural radioactivity, necessitate that practical limits include the concentrations normally found in the majority of houses.

A simple first approach toward limiting exposure to radon from water sources would be to use the historic, conservative "tolerance doses" of 1/10 to 1/100--the exposures known to affect health. (For a discussion of this concept, see Cr74). The reduction factors are presumably dependent on the percent incidence or severity of effect. If we assume that indoor radon and radon-daughter exposures have an associated health effect, and further assume that average indoor radon levels from all sources, including drinking water, are on the order of 1 pCi/L (NAS81), the "tolerance" inhalation exposures, per se, are 0.01 to 0.1 pCi/L. (Note: we do not propose to limit total indoor radon concentrations to these levels, the calculation is simply for illustration.) Based on the mean transfer coefficients of $1 \text{ to } 2.5 \times 10^{-4}$ for radon in air from radon in water, the derived concentration limit for radon in water ranges from 40 to 1000 pCi/L [(0.01 to 0.1 pCi/L) $\div$ ($1 \text{ to } 2.5 \times 10^{-4}$)], if all of the indoor radon were to come from the water supply. For houses containing radon in the water supply, 5 to

12% of indoor radon, on average, has been estimated to come from the water (Ka80; UN81). In our calculations, however, we generally assume that all indoor radon comes from the water supply. Because the health hazard is expected to be low for average indoor radon exposures, an upper rounded value of 1000 pCi/L radon in water is the more reasonable choice.

An even simpler approach would be to assume that the risk associated with average indoor concentrations of radon and radon daughters is acceptable to the U.S. population. The possibility that this risk may be less than the linear hypothesis predicts, or may even be zero, lends some support to this assumption. Under this assumption, the derived concentration limit for radon in water ranges from 4000 to 10,000 pCi/L [1.0 pCi/L ÷ (1 to 2.5 x $10^{-4}$)], if all the indoor radon were to come from the water supply.

Another example of the "tolerance dose" approach is to assume that lifetime exposures above about 100 WLM have a statistically significant excess of lung cancer and that exposures below this level have only a low probability for causing deleterious health effects. This choice is based on the epidemiological review by the NCRP (NCRP84a) which also concludes that none of the studies, thus far, have produced data showing a statistically significant excess of lung cancer in the lowest exposure category (<60 WLM). Applying the tolerance factor of 1/10, the lifetime exposure limit is 10 WLM, a value comparable to average lifetime exposures to environmental radon. The derived radon concentration in air for an average indoor radon-daughter equilibrium factor of 0.5 and an exposure period of 60 year, is 0.65 pCi/L [10 WLM x 170 WLhr/WLM x 1/8760 yr/hr x 1/60 yr$^{-1}$ x

1/0.5 x 100 pCi/L/WL]. On the assumption that all of the indoor radon comes from the water supply, the derived concentration limit for radon in water ranges from 2600 to 6500 pCi/L.

In summary, these three approaches suggest that the upper limit on radon in water, based on the conservative "tolerance dose" approach; continuous inhalation exposure; and the assumption that 100% of the radon in indoor air comes from the water supply, is a rounded value between 1000 and 10,000 pCi/L.

An alternative, more fruitful approach would be to base risk on the distribution of population exposures to natural radioactivity (NCRP84b); basing risk on fractions of the dose equivalents allowed for radiation workers is tenuous at best, because we really do not know the true risk associated with occupational dose equivalents. If we know the average exposure, and the range that a population receives, a practical upper limit on exposure could be established on a cost-risk-benefit, remedial-action basis. It is unlikely that this approach could be rigorously pursued at this time, however, due to the paucity of data regarding population exposure to radon and radon daughters in the United States.

The distribution approach has been taken in Canada, confirming an earlier indoor limit of 0.02 WL (1 WLM/yr) for uranium mining communities (Ea82). If we apply this radon-daughter concentration to all communities in the U.S. and assume that the average, indoor, radon-daughter equilibrium factor is 0.5, a derived upper limit on indoor radon air-concentrations is 4 pCi/L [0.02 WL x 100 pCi/L/WL x 1/0.5]. The derived upper concentration limit on radon in water (assuming all radon comes from the water) would range from 16,000 to 40,000 pCi/L. The actual value would depend on the amount of radon coming from soil gas and other sources. It is expected that the average risk for all U.S. residents would be lower than the maximum risk associated with the 4 pCi/L limit on indoor air concentration.

While the actual U.S. population exposure distribution is unknown, the estimate of the mean indoor exposure is 0.2 WLM/yr for first-floor exposures in houses (Har81). The average annual exposure for all members of the U.S. population may be no greater than about 0.13 WLM/yr, considering that a significant fraction of the population resides in multistory buildings (Har81). Tables 5 and 6 show estimated distributions of the lifetime risk for average annual exposures of 0.2 and 0.13 WLM, respectively, assuming lognormal exposure distributions and a geometric standard deviation (GSD) of 2.5. A GSD of about 2 is in keeping with the values measured in New York and New Jersey residences (Ge80); however, the measurements by McGregor et al. (McG80) in Canadian houses, and by Prichard et al. in Maine and Texas houses (Pr81), indicate that mean GSD values are closer to 2.5.

Tables 5 and 6 indicate that the majority of the risk occurs in the lower exposure categories. Thus, any limits on exposure would have to take this fact into consideration. The lower of the two distributions (0.13 WLM/yr, Table 6) indicates that only a very small percentage (<0.03%) of the population has an exposure to radon daughters exceeding 2 WLM/yr, whereas approximately 0.35% is exposed to levels exceeding 1 WLM/yr. For populations exposed to an estimated average of 0.2 WLM/yr radon daughters (Table 5), approximately 0.14% receive exposures exceeding 2 WLM/yr, while 1.3% exceed 1 WLM/yr. In either case, an exposure limit of 2 WLM/yr would require relatively little remedial action, whereas an exposure limit of 1 WLM/yr would necessitate remedial action on about 10 times more houses.

The tables also indicate that substantial breaks in the lung-cancer risk occur at the 1-WLM/yr and 2-WLM/yr exposures. This suggests, in the absence of cost-risk-benefit analyses, that either exposure limit might be reasonably selected on a practical basis. Limits less than 1 WLM/yr appear to require remedial action in an impractical number of houses.

If we again choose the 1-WLM/yr exposure limit as an illustration, the derived, allowable, radon-air concentration (assuming an equilibrium factor of 0.5 and continuous exposure) becomes 4 pCi/L, a value approximately four times higher than the estimated average indoor level in U.S. houses (NAS81). This allows, on average, approximately 3 pCi/L to come from water. The average, derived, radon level in water sources, therefore, ranges from about 10,000 to 30,000 pCi/L [3 pCi/L $\div$ (1 to 2.5 x $10^{-4}$)]. Had 2 WLM/yr been picked as the indoor annual limit on exposure, the derived, allowable radon concentration in water would have ranged between about 20,000 and 60,000 pCi/L. For those houses where all of the indoor radon comes from the water supply, the derived upper concentration limits on radon in water range from 16,000 to 40,000 pCi/L for the 1-WLM/yr inhalation exposure limit and 32,000 to 80,000 pCi/L for the 2-WLM/yr inhalation exposure limit.

Finally, an equivalent-risk approach could be used to derive the concentration limit for radon in water. Based on the risk factors in Table 3, 20,000 pCi/L $^{222}$Rn in water has an associated lifetime, total, cancer-death risk ranging from 8 to 16 x $10^{-3}$ [20,000 pCi/L x (4 to 8 x $10^{-7}$

cancer deaths/pCi/L)], or approximately 1 to 1.5%. The associated lifetime, lung-cancer-death risk ranges from 0.6 to 1.5%. These lifetime risks are comparable, by our calculation, to the lifetime lung cancer risk presently allowed by the EPA in their standards issued for cleanup of uranium tailings (FR83). Under EPA's standard for buildings, the objective is to achieve an indoor radon-daughter concentration of 0.02 WL. Tailings are to be removed from premises where the levels exceed 0.03 WL, but lower-cost ventilation and air cleaning methods may be employed instead. The allowable 0.02 to 0.03 WL concentrations have an associated lifetime, lung-cancer-death risk of 1 to 1.5%, assuming continuous exposure and a risk-coefficient of $10^{-2}$ per WLM/yr. For comparison, the EPA values for 0.02 WL lifetime environmental exposures are 1 and 2.3%, respectively, based on their absolute and relative risk models (RPC80).

Based on these considerations of the estimated distribution of radon exposures in the U.S., a derived practical limit on radon concentrations in water is not less than 10,000 pCi/L. A 20,000-pCi/L value is reasonable and conservative from the standpoints of limiting cost of remedial action to a more manageable number of houses; the exposure is considered to be continuous from infancy; the value is based on an air-water transfer coefficient high enough to accommodate reasonable energy-conservation measures; and it is based on an assumed, average equilibrium factor of 0.5 for daughters derived from the radon released from water. The derived water concentration would be higher for equilibrium factors <0.5. In houses where the total radon-daughter equilibrium factor is substantially lower than about 0.5, it is almost certain that the radon concentration limit could be higher. Until more data are available regarding the actual exposure distribution in the U.S., the 20,000 pCi/L choice is considered to be the best balanced estimate, in the absence of cost-risk-benefit analyses, of the derived radon-concentration limit for water.

We do not wish to emphasize the radon in water concentration per se, but rather the primary inhalation exposure limit in WLM/yr. Based on estimated, natural exposure distributions in dwellings, we suggest that a limit on exposure less than 1 WLM/yr (including background exposures) might not be reasonable from a cost-benefit perspective. Some might argue this to be the case even at 2 WLM/yr. Whatever choice is made for exposures applicable to all buildings (not just tailings-contaminated buildings), we believe that the primary emphasis regarding remedial action should be placed on an annual limit on total inhalation exposure (WLM/yr) and not on a derived WL-concentration of radon daughters or a derived air concentration of radon gas. Keeping the emphasis on the primary standard would allow for variability in occupancy time, ventilation rate, and other factors bearing on the exposure of individuals occupying a structure. The derived limit on radon in water (20,000 pCi/L) should, therefore, be looked on as an action level, above which consideration would be given to characterizing the indoor radon-daughter exposures. Experience may eventually allow adjustment of this derived concentration to a much higher level.

Finally, it should be pointed out that, based on the previous estimates of dose to stomach and whole body, a 20,000-pCi/L radon concentration in water would produce estimated annual dose-equivalents to stomach and whole body of 2 rem and 0.04 rem, respectively. While the current NCRP population limits on annual whole-body dose (0.17 rem/yr) are not exceeded at this concentration (and would not be exceeded at about 4 times this concentration), the dose to the stomach is higher than normally accepted for population exposures. Four caveats are in order when discussing these doses. First, the NCRP is restudying the whole issue of population limits regarding exposures to manmade sources; thus, our comparison base may change. Second, exposures very often exceed the population limits, which are based on a factor of 10 to 30 reduction of worker exposures. One might convincingly argue that the population limits on exposure are unduly conservative when compared with certain natural-background exposures. Third, the radiation risk to stomach is significantly lower than that to lung. This fact is based on the lack of supportive evidence of stomach cancer in workers exposed to high levels of radon and radon daughters (such as underground miners) and the additional negative evidence in the animal experiments. Fourth, the evaluation of ingestion doses by Sullivan and Nelson, discussed above, indicates that the stomach-dose estimates used in this paper are unduly conservative. It appears at this time, therefore, that a dose limitation approach based on the stomach dose would be unrealistic for deriving allowable radon concentrations in water.

In summary, the derived upper radon concentration in water (assuming all indoor radon comes from the water supply) ranges from 1000 to 10,000 pCi/L, based on continuous exposure and the conservative "tolerance dose" approach; 16,000 to 40,000 pCi/L, based on continuous exposure and the exposure-distribution approach, and the 1-WLM/yr inhalation exposure limit; and 32,000 to 80,000 pCi/L based on the exposure-distribution approach and the 2-WLM/yr inhalation exposure limit.

We suggest by our analysis that a rounded concentration value of 10,000 pCi/L can be supported by health-effects considerations alone, based on the "tolerance-dose" concept and other conservative assumptions regarding dose. We further suggest that a practical value of not less than 10,000 pCi/L can be supported by the estimated distribution of environmental radon-daughter exposures in the U.S. The suggested 20,000-pCi/L concentration limit (or action level) has an estimated lifetime cancer-death risk comparable, by our calculation, to current EPA standards for uranium-tailings-contaminated buildings.

Research needed for resolution of the uncertainties in the values discussed is primarily related to improving the estimations of the inhalation, rather than the ingestion, exposures and doses. Major examples are: improved exposure-distribution data for the U.S. population; realistic values for household occupancy patterns; more accurate data on radon-daughter equilibrium factors; more accurate data on the ratio of water-contributed radon to all other sources of indoor radon; more accurate data on the water-to-air transfer coefficient; and more accurate cancer-risk factors.

Finally, we would like to emphasize that, before a maximum contaminant level (MCL) for radon in water can be firmly established, the broader issue of the MCL for radon in indoor air must be addressed.

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TABLE CAPTIONS

Table 1. Lifetime Lung-Cancer Risk Coefficients for Lifetime Environmental Exposures

Table 2. Risk-Coefficient Adjustment Factors vs Radon-Daughter Disequilibrium

Table 3. Summary of Cancer Risks per pCi/L Radon Concentration in Drinking Water

Table 4. Lifetime Cancer-Risk Versus Radon Concentration in Water

Table 5. Calculated Distribution of Lung-Cancer Risk for Populations Exposed to an Average of 0.2 WLM/yr Radon Daughters (GSD, 2.5)

Table 6. Calculated Distribution of Lung-Cancer Risk for Populations Exposed to an Average of 0.13 WLM/yr Radon Daughters (GSD, 2.5)

Table 1. Lifetime Lung-Cancer Risk Coefficients for Lifetime Environmental Exposures(a)

| Age at First Exposure | Exposure Units | |
|---|---|---|
| | Per WLM/yr | Per pCi/L |
| 1 yr | $9.1 \times 10^{-3}$ | $3.6 \times 10^{-3}$ |
| Mixed(b) | $5.6 \times 10^{-3}$ | $2.1 \times 10^{-3}$ |

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(a) Radon daughter equilibrium factor = 0.71, unattached RaA/Rn ratio = 0.07.

(b) Pertains to populations with ages characteristic of U.S. inhabitants in 1975.

Table 2. Risk-Coefficient Adjustment Factors vs Radon-Daughter Disequilibrium

| Radon and Radon Daughter Ratios | Equilibrium Factor[(a)] | Adjustment Factor for Radon-Daughter Risk Coefficient | Adjustment Factor for Radon-Gas Risk Coefficient |
|---|---|---|---|
| 1/0.9/0.7/0.7 | 0.71 | 1.00 | 1.00 |
| 1/0.9/0.6/0.4 | 0.55 | 1.05 | 0.81 |
| 1/0.6/0.3/0.2 | 0.29 | 1.30 | 0.53 |
| 1/0.256/0.098/0.084 | 0.11 | 2.21 | 0.34 |

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(a) The equilibrium factor is the ratio of the total potential alpha energy of the actual, short-lived daughter concentrations to the total potential alpha energy that the daughters would have if they were in equilibrium with radon.

Table 3. Summary of Cancer Risks per pCi/L Radon Concentration in Drinking Water

| Organ | Lifetime Risk | Annual Risk[b] |
|---|---|---|
| Lung[a] | $3 \text{ to } 7.5 \times 10^{-7}$ | $7 \text{ to } 17 \times 10^{-9}$ |
| Stomach | $<6 \times 10^{-8}$ | $<1 \times 10^{-9}$ |
| Whole Body | $1 \times 10^{-8}$ | $2 \times 10^{-10}$ |

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(a) Lung cancer risks are derived from the lung-cancer-prediction model.

(b) Assuming 45 years at risk for lung cancer and 60 years at risk for stomach and whole-body cancer deaths.

Table 4. Lifetime Cancer-Risk Versus Radon Concentration in Water

| Radon Concentration (pCi/L) | Lifetime Risk (%) |
|---|---|
| 5,000 | 0.2 to 0.4[a] |
| 10,000 | 0.4 to 0.8 |
| 15,000 | 0.6 to 1.2 |
| 20,000 | 0.8 to 1.6 |
| 25,000 | 1 to 2[b] |

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[a] Equivalent to estimated lifetime risk from estimated average indoor concentrations (1 pCi/L) in houses.

[b] Equivalent to estimated lifetime risk of occupational standard of 4 WLM/yr for 30 yr.

Table 5. Calculated Distribution of Lung-Cancer Risk for Populations Exposed to an Average of 0.2 WLM/yr Radon Daughters (GSD, 2.5)

| WLM/yr Range | % of Population | Fractional Lifetime Risk(a) | Fractional Annual Risk | Annual Deaths per $2.2 \times 10^8$ Persons(b) |
|---|---|---|---|---|
| 0 - 0.2 | 68 | $6.0 \times 10^{-4}$ | $1.3 \times 10^{-5}$ | 2919 |
| 0.2 - 0.5 | 25 | $7.9 \times 10^{-4}$ | $1.8 \times 10^{-5}$ | 3865 |
| 0.5 - 1.0 | 5.7 | $4.0 \times 10^{-4}$ | $9.0 \times 10^{-6}$ | 1970 |
| 1.0 - 2.0 | 1.2 | $1.7 \times 10^{-4}$ | $3.8 \times 10^{-6}$ | 830 |
| 2.0 - 4.0 | 0.14 | $4.0 \times 10^{-5}$ | $8.8 \times 10^{-7}$ | 194 |
| TOTAL | 100 | $2.0 \times 10^{-3}$ | $4.4 \times 10^{-5}$ | 9778 |

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(a) The attributable risk from radon (radon-daughter) exposures can be compared with the present lifetime risk of lung cancer [about $4 \times 10^{-2}$ in the U.S., according to Evans et al. (Ev81)], which is largely attributable to cigarette smoking.

(b) Ten percent of the risk occurs above 1 WLM/yr; 2% occurs above 2 WLM/yr.

Table 6. Calculated Distribution of Lung-Cancer Risk for Populations Exposed to an Average of 0.13 WLM/yr Radon Daughters (GSD, 2.5)

| WLM/yr Range | % of Population | Fractional Lifetime Risk[a] | Fractional Annual Risk | Annual Deaths per $2.2 \times 10^8$ Persons[b] |
|---|---|---|---|---|
| 0 - 0.13 | 68 | $3.3 \times 10^{-4}$ | $7.4 \times 10^{-6}$ | 1630 |
| 0.13 - 0.5 | 29.3 | $7.5 \times 10^{-4}$ | $1.7 \times 10^{-5}$ | 3652 |
| 0.5 - 1.0 | 2.35 | $1.7 \times 10^{-4}$ | $3.7 \times 10^{-6}$ | 812 |
| 1.0 - 2.0 | 0.322 | $4.6 \times 10^{-5}$ | $1.0 \times 10^{-6}$ | 223 |
| 2.0 - 4.0 | 0.028 | $7.9 \times 10^{-6}$ | $1.8 \times 10^{-7}$ | 39 |
| TOTAL | 100 | $1.3 \times 10^{-3}$ | $2.9 \times 10^{-5}$ | 6356 |

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[a] The attributable risk from radon (radon-daughter) exposures can be compared with the present lifetime risk of lung cancer [about $4 \times 10^{-2}$ in the U.S., according to Evans et al. (Ev82)], which is largely attributable to cigarette smoking.

[b] Four percent of the risk occurs above 1 WLM/yr; 1% occurs above 2 WLM/yr.

## COMMITTEE ON OCCURANCE

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Jacqueline Michel
Howard M. Pritchard

# THE OCCURRENCE OF RADIOACTIVITY IN PUBLIC WATER SUPPLIES IN THE UNITED STATES

C. T. Hess
University of Maine
Department of Physics, Orono, Maine 04469

J. Michel
Research Planning Institute, Inc.
925 Gervais, Columbia, South Carolina 29201

T. R. Horton
Eastern Environmental Radiation Facility
United States Environmental Protection Agency
P.O. Box 3009, Montgomery, Alabama 36193

H. M. Prichard
University of Texas School of Public Health
P.O. Box 20186, Houston, Texas 77025

W. A. Coniglio
Office of Drinking Water
United States Environmental Protection Agency
401 Street, S.W., Washington, D.C. 20460

## ABSTRACT

Examination of the collected data for radionuclide concentration measurements in public water supplies in the United States show more than 51,000 measurements for gross alpha particle activity and/or Radium, 89,900 measurements for uranium, and 9,000 measurements for radon. These measurements were made as part of national and state surveys of radionuclide concentrations in utility water supplies for radium and radon; and the NURE survey for uranium which included non-utility water supplies.

Surface water has low values for radium and radon but levels comparable to ground water for uranium. Separate isotope measurements were not taken for much of the radium and uranium data. Because $^{226}$Ra to $^{228}$Ra ratios and $^{238}$U to $^{234}$U ratios are not fixed in water, further measurements are needed to establish the specific isotopic concentrations by region. Analysis of the state average values in geological provinces show the highest provincial areas for radium are the Upper Coastal Plain, glaciated Central Platform, and Colorado plateau. For uranium, the highest areas are Colorado plateau, West Central Platform, and Rocky Mountains. For radon, the highest provinces are New England and the Appalachian Highlands - Piedmont. Regional hydrogeological and geochemical models are suggested for guiding the formulation of regional standards and monitoring strategies. Utility supplies serving small populations have the highest concentration for each radionuclide and have the lowest fraction of samples measured, which shows a need for further measurements of these small population water supplies. Risk estimates for the average concentration of radium in utility ground water give about 2700 fatal cancers per 70.7 year lifetime in the United States. Risk estimates for the average concentration of uranium in utility surface and ground water give about 600 fatal cancers per 70.7 year lifetime in the United States. Using 1 pCi/l in air for 10,000 pCi/l in water, the radon in utility water risk estimate is for 4,400-22,000 fatal cancers per 70.7 year lifetime in the United States.

## PREFACE

The sections of this paper are arranged in the order of introduction, geochemistry and occurrence. A central theme of all sections of the report is that the geological setting strongly controls the occurrence of natural radionuclides in drinking water. The observed concentrations of U, Ra, and Rn in ground and surface water can be related to the rock types and the amount and distribution of U and Th in the materials which constitute the aquifer and surficial deposits. The United States can be divided into eleven geological provinces, each of which is characterized by dominant types of rocks or deposits as well as ground water flow systems, discussed in Table 1 and shown in Figure 1. (Be81, Sc62). These provinces are discussed in all sections of the report and provide a framework for understanding the variations in the distribution and activities of natural radionuclides in water. In fact, one hypothesis is that certain provinces or sub-provinces can be characterized as producing ground water with specific radionuclide problems, or conversely, without specific radionuclide problems. If this hypothesis can be verified, it has important applications to the development of regional guidelines for monitoring requirements in the revised regulations (La83).

# OCCURRENCE OF RADIUM ISOTOPES IN PUPLIC DRINKING WATER

## INTRODUCTION

Radium has two natural isotopes which are of concern in public water supplies. Radium-226 (Ra-226) is generated through decay of uranium-238 (U-238) and is an alpha emitter with a $t_{1/2}$ = 1,622 years. This is the isotope which is commonly referred to as radium and has been measured in many water supplies. The other isotope, Ra-228, is generated directly by thorium-232 (Th-232) decay and is a shorter-lived, weak-beta emitter ($t_{1/2}$ = 5.7 years). There is a third isotope of radium which is of possible concern, Ra-224 with a $t_{1/2}$ = 3.64 d. Its occurrence is not well known, only a few data are available from samples at the well head. The U.S. Environmental Protection Agency (EPA) established interim regulations in 1976 for maximum levels of radioactivity in drinking water as follows:

> Maximum contaminant levels (MCL) of combined Ra-226 and Ra-228 - 5 picocuries per liter (pCi/l); gross alpha-particle activity - 15 pCi/l excluding radon and uranium. (Ep76a).

These MCLs were set under the authority of the Safe Drinking Water Act to protect health, taking treatment costs in consideration. In an effort to minimize the costs of analysis and monitoring, EPA established a series of screening steps to test for compliance with the interim regulations. These criteria stated that when the average gross alpha-particle activity of four quarterly samples or composites exceeds 5 pCi/l, the same or equivalent sample shall be analyzed for Ra-226. If the activity of Ra-226 exceeds 3.0 pCi/l, the sample shall be analyzed for Ra-228. Inherent in these regulations were the assumptions that Ra-226 was to be the dominant radioactive contaminant in drinking water and the Ra-228/Ra-226 activity ratio was less than 1.0. The regulations required all systems supplying 25 or more people to be monitored every four years.

Since the interim regulations were established, much more information on the occurrence of radium isotopes is now available from state compliance data and from detailed studies on the correlation and interrelationships of Ra-228 and Ra-226 in ground water with specific geological provinces ((Mi80), (As81), (Ki82), (Mi82), and (Kris82)). In light of these new data, the key issues to be considered for revision of the regulations are:

1) Prioritization of specific areas for monitoring for Ra-228 and Ra-226;

2) Reduction in the interval frequency or complete omission for specific areas for repeat monitoring; and

3) Decoupling Ra-228 analysis from Ra-226, with criteria for when Ra-228 is to be measured.

The purpose of this paper is to concisely review the existing information on the geochemistry and occurrence of Ra-228 and Ra-226, and to provide guidelines for regulatory revision.

## GEOCHEMISTRY OF RADIUM ISOTOPES

The distribution of Ra-228 and Ra-226 in water is a function of the thorium and uranium content of the aquifer, the geochemical setting of the aquifer solids, and the $t_{1/2}$ of each isotope. There are specific geological and chemical processes that control the thorium and uranium content in aquifers, which are discussed in detail by Olson and Overstreet (0164), Cherdynstev (Ch71), and Gableman (Ga77). In fact, thorium and uranium have very similar behavior, with one important exception which is most responsible for their eventual separation. Thorium has one oxidation state and is immobile at low temperatures. Therefore, thorium distribution is controlled by primary geochemical processes (such as magmatic crystallization) or secondary physical processes (such as sedimentary enrichment in placer deposits). Uranium has two oxidation states and the +6 state (uranyl) can form highly soluble complexes which can be transported long distances by oxidizing ground water before being removed by adsorption or reduction to the +4 state. The estimated average crustal Th/U activity ratio is 1.2-1.5 so that, in the absence of enrichment or depletion processes, Ra-228 activity should be higher than Ra-226. However, the tendency for uranium enrichment under certain geochemical conditions results in regions of higher Ra-226, thus EPA's decision to emphasize Ra-226 in the interim regulations.

Radium enters ground water by dissolution of aquifer solids; by direct recoil across the liquid-solid boundary during its formation by radioactive decay of its parent in the solid (both isotopes have thorium as the immediate parent), and by desorption. The mechanism of alpha recoil is an important factor in the higher solubility of daughter isotopes compared with their parents. U-234/U-238 activity ratios in ground water are generally greater than 1.0 and can be as high as 28 (Gi82). Ra-224/Ra-228 activity ratios in South Carolina ground water range from 1.2 to 2.0 (W. S. Moore, unpubl. data) and in Connecticut from 0.8 to 1.7 (Kris82). However, when the daughter/parent pair consists of different elements, geochemical factors become important controls of their relative solubility. An extreme example is Rn-222, the immediate daughter of Ra-226; Rn-222/Ra-226 activity ratios in water can be as high as $10^6$. Because of alpha recoil and the different solubilities of the thorium and uranium series isotopes, extensive disequilibrium occurs in ground water.

Recent studies have suggested that radium is rapidly absorbed from ground water. King et al. (Ki82) proposed that the distance of Ra transport in ground water was less than that of Rn-222 (with a $t_{1/2} = 3.8$ days) due to continual adsorption of radium onto the aquifer solids. Krishnaswami et al. (Kris82) calculated adsorption and desorption rate constants for radium in Connecticut aquifers and proposed that radium removal rates are rapid, as short as a few minutes. Equilibrium between adsorption and desorption is also quickly established, but Krishnaswami et al. concluded that the partition coefficient strongly favors the solid phase, and almost all radium introduced into the ground water studied resides on particle surfaces in the adsorbed state. However, the extent of sorption is controlled by the geochemical reactivity of the aquifer material. King et al. (Ki82) notes that the average Ra-228 and Ra-226 activity in the crystalline aquifers of South Carolina was lower than for the Coastal Plain sediments, even though the thorium and uranium content of the rock aquifers was higher. Furthermore, the Rn-222 activity in the crystalline aquifers was ten times greater than the aquifers sampled in the Coastal Plain. King et al. concluded that the affinity of radium for adsorption sites in the fresh rock surfaces which have higher cation-exchange capacities was greater than for the sand and gravel deposits composed of refractory minerals such as quartz. Thus, radium in ground water does not accumulate with ground water transport in aquifers; it stays very close to the area in which it is produced.

The insolubility of radium and thorium can be inferred from studies of potential contamination of ground water due to seepage from uranium tailings ponds in New Mexico reported by Kaufmann et al. (Ka76). At one such pond, they estimated that nearly 3 x $10^9$ L of seepage entered the shallow aquifer over a 20-year period. The wastes in this pond contained approximately 200 pCi/l of Ra-226 and 166,000 pCi/l of Th-230. Thus, nearly a Ci of Ra-226 and 500 Ci of Th-230 were available to leach with the shallow ground water; yet, in 1975, monitoring wells located 1 km down-gradient from the pond showed no evidence of contamination.

Through an understanding of the physical and chemical processes which control radium distribution, we can now begin to interpret the new data base from state compliance reports, and to develop predictive models for radium occurrence on which new regulations should be structured. These proposed models would characterize certain geological settings or aquifer types as producing ground water with high or low radium content. EPA has begun to develop a predictive model for the occurrence of Ra-228, with a pilot study completed for two geological provinces, the Atlantic and Gulf Coastal Plain sedimentary aquifers and the Piedmont rock aquifer of the eastern United States (Mi82). Information on areas of high radium occurrence is necessary to provide guidance to states for additional monitoring. From a regulatory point of view, areas of low radium activity are very important, in that they could have a different monitoring priority and schedule. A predictive model for Ra-228 would also be valuable because so few samples were measured under the present analytical scheme.

## OCCURRENCE OF Ra-226 AND Ra-228 IN DRINKING WATER

All but six states (Illinois, Nebraska, Colorado, Utah, Montana, and Oregon) have reported known MCL violations for radium as required by the interim regulations. There are approximately

200 reported public water suppliers with Ra-226 activities in excess of 5 pCi/l after normal treatment (Co, in prep.). The following sections discuss these results and other studies by water types, geological setting, and isotope.

## Surface Water

The radium content of surface water is usually very low. Ra-226 generally ranges between 0.1 and 0.5 pCi/l and the Ra-228/Ra-226 activity ratio is generally greater than unity (Mo69), (El83). Also, standard water treatment methods are known to remove radium (Ep76b). To the best of our knowledge, no surface water violations for radium have been reported by the states. Thus, surface water systems should be separately evaluated, perhaps they could be released from monitoring requirements for radium once the source stream was documented as having low natural radioactivity.

## Ground Water

Out of the nearly 60,000 public water supplies in the United States, about 80 percent use ground-water sources. Over 90 percent of the ground-water supplies serve less than 3,300 people and are classified as small or very small. In general, radium in drinking water is a small-system problem. Figure 2 is a compilation of the areas and specific sites which have high radium in ground water from both state compliance data and published studies.

From the data reported by the states the mean total radium activity for supplies exceeding the MCL was almost 10 pCi/l. Ra-226 activity was generally greater than Ra-228 activity, but these data were initially biased toward high Ra-226. King et al. (Ki82) found that the average Ra-228/Ra-226 activity ratio was 1.2 for over 180 samples throughout South Carolina. Of these, ten samples had Ra-226 less than 3 pCi/l, but total radium greater than 5 pCi/l. King et al. (Ki82) estimated that perhaps 40-50 percent of the total radium violations for the Piedmont and Coastal Plain provinces were missed using the prescribed screening procedure which couples Ra-228 analysis to Ra-226. Kriege and Hahne (Krie82) reported additional sampling which identified eight violations for total radium although the Ra-226 was less than 3 pCi/l.

From the available data, there are two specific geological regions where over 75 percent of the known radium violations occur:

1) The Piedmont and Coastal Plain provinces in New Jersey, North Carolina, South Carolina, and Georgia; and
2) A north-central region, consisting of parts of Minnesota, Iowa, Illinois, Missouri, and Wisconsin.

The rest of the violations are generally scattered clusters, notably along the Arizona-New Mexico border, Texas, Mississippi, Florida, and Massachusetts (Fig. 2). All of these scattered violations had high Ra-226 activities, as would be expected from the screening methods used to detect them. Ra-228 activities in these systems were very low. We believe that the current analytical protocol has detected a large percentage of the systems with high Ra-226. Cothern and Lappenbusch (Co84) have used the compliance data for Ra-226 to estimate that approximately 500 systems will be determined to exceed the MCL of 5 pCi/l.

Improvement on this estimate is difficult with the existing data base, which is comprised mostly of reported MCL violations for Ra-226. Statistical analysis of these data is not possible because they were not randomly sampled. In this respect, states should be requested to submit all radium results to facilitate further analysis. However, some calculations can be made to corroborate the previous estimates of MCL violations. Data from South Carolina (Ki82) showed that approximately 3.0 percent of the ground water supplies exceeded the 5.0 pCi/l limit for total radium. (Note that the prescribed screening procedures detected only one-half of these violations). Applying that percentage to North Carolina and Georgia, both of which have similar hydrogeology, provides an estimate of 150 violations for all three states. In Iowa, approximately 10 percent of the 605 supplies sampled to date, using a lower screening criteria, exceed the MCL. Again, applying this percentage to all the ground water systems of Iowa and half of Illinois, Missouri and Wisconsin yields 120 violations for Iowa, 75 violations for Illinois and 50 for Missouri, and 60 for Wisconsin. We can estimate violations for the states that have not reported as follows: 10 each for Utah, Colorado, and Nebraska, and zero for Oregon and

Montana. There are 71 violations reported in all other states. The total of these known and estimated violations is 556. Assuming 10-25 percent of the actual violations are missed during the prescribed screening procedure (actual data for Iowa, 8 out of 60 or 13 percent; for South Carolina, 8 out of 30 or 26 percent), the number of violations ranges between 600 and 700.

Estimates of population exposure nationwide can only be broadly made without additional information on populations served by the MCL violations, as well as on the results of all analyses. Lucas (Lu82) estimated that 91 communities in Illinois, Iowa, Missouri, and Wisconsin with a population of 599,000 consume water with Ra-226 greater than 5.0 pCi/l.

Under the present screening methods, however, there was concern that Ra-228 violations were not being detected. Thus, EPA recently funded a study to determine if a predictive model for the occurrence of Ra-228 could be developed. The Piedmont and Coastal Plain aquifers were selected as a pilot study area for development of a model because the radiochemistry of these provinces had been extensively studied, these were areas of known high Ra-228 activities, and nearly 300 values for Ra-228 were available.

The model comprises a multilevel classification of aquifer characteristics for each Ra-228 datum. The nature of the data precluded use of such analytical tools as regression analysis to establish a quantitative relationship between, for example, thorium content and Ra-228. As a result, the underlying model structure was evaluated to assess the existence of differences not only between the major aquifer types but also between lower-level classification within a major aquifer type. A detailed description of the model development, parameters, methodology, and results can be found in a report by Michel and Pollman (Mi82). Only a summary is discussed below. Also, although the model was developed specifically for Ra-228, values for Ra-226 were available and similar statistics were calculated. There is much to be learned from these differences in the results for these two isotopes.

Table 2 summarizes the means and ranges for those aquifer types which had significantly different Ra-228 distributions. Note the striking differences in the means, although the ranges are similar in some cases. Arkosic (immature, feldspar-rich) sand aquifers had mean values for both radium isotopes up to an order of magnitude greater than quartzose sands. Limestones and metamorphic rock aquifers in the study area had very low activities of both radium isotopes. Table 3 shows a ranking of all structural levels used in the pilot study, with symbols indicating groups of similarity of Ra-228 distribution (with Ra-226 means are also given without a ranking). Classes identified by the same letter code in the grouping column represent subsets of a group that are statistically indistinguishable from other classes of the same group. However, groupings that overlap indicate that the particular individual groups are not unique. For example, group A as a class is not statistically different from groups B, C, and D; groups E, F, and G, however, represent groups of classes with significantly lower Ra-228 activities than group A. Although some groups were not significantly different, the ranking followed the anticipated trends within major types. For example, the ranking of igneous rocks, with syenite>granite>diorite, follows thorium abundance in these rock types. Arkosic sand aquifers are ranked in order of thorium content of the source rock, from high to low. This pilot study showed that specific aquifer types and geochemical conditions can be characterized as producing ground water with high or low Ra-228 activities. Its application can be demonstrated for the aquifers of the Piedmont and Atlantic and Gulf Coastal Plain provinces; high Ra-228 was likely to occur in aquifers composed of (1) acidic, igneous rocks and (2) arkosic sands with sources having high-to-medium thorium content.

The results from the model were used to map specific areas (aquifer types), from New Jersey to Alabama, that would be likely to produce ground water with high Ra-228. In fact, in the Piedmont province, granitic rock aquifers younger than 350 million years were shown to produce high-radium ground water. Older rocks had undergone metamorphism which has tended to recrystallize thorium and uranium into resistate minerals in which radium is more tightly bound. The arkosic sand aquifers were restricted to the upper Coastal Plain from Virginia to Georgia. These aquifers are composed of sediments eroded from the nearby Piedmont rocks and are mineralogically immature. They contain higher amounts of thorium- and uranium-bearing minerals than the middle and lower Coastal Plain sediments which were deposited farther from the source rocks. Thus, the trend in the Coastal Plain aquifers for Ra-228, whose parent is not subject to secondary transport processes, is one of decreasing activities with distance from the Piedmont source. Only 1 out of 50 samples from the quartzose sands of the middle and lower Coastal Plains aquifers was greater than 3 pCi/l for Ra-228. In contrast, Ra-226 in the middle Coastal Plain aquifer is highly variable, with values from 0 to 196 pCi/l, due to the ability of its parent to migrate in ground water and undergo secondary enrichment.

Knowledge of the conditions where very low radioactivity will occur is also very important. In the area studied, low Ra-228 occurred in aquifers of (1) metamorphic rocks, (2) quartzose sands, and (3) limestone. Thus, the lower Coastal Plain, composed of extensive limestones and deep quartzose sand aquifers, is notable for its total lack of Ra-228 greater than 1.0 pCi/l in ground water. Ra-226 will be more variable because of the high solubility of uranium complexes in the carbonate system, but it is generally detected by the gross-alpha particle activity screen. Nevertheless, there have been only three Ra-226 violations reported for the entire lower Coastal Plain province, from New York to Texas. These violations were all from one region, in Florida. The second area is the North-Central region. Much of the ground water comes from deep aquifers, frequently having Ra-226 activities of 5-25 pCi/l; Ra-228 can be as high as 32 pCi/l (Lu82). There is no apparent correlation between Ra-228 and Ra-226 and no specific trends in their distribution by aquifer, depth in the aquifer, or areal extent. Interpretation of the radium distribution in this area is complicated by complex hydrogeology and multiply screened wells in different aquifers. There is evidence of significant uranium migration, both during geological time and the present, which provides a mechanism for high Ra-226 as well as resulting in a complex distribution and disequilibrium of uranium series isotopes (Gi82);(Li02). Possible sources for high Ra-228 have not been identified, but the Ra-228 distribution may be able to be explained by analysis of the sources, depositional setting, and diagenesis of the sedimentary rock aquifers.

Limited work has been done on radium occurrence in the other geological provinces. Ra-226 has been found to be high in areas of uranium mineralization, such as in Texas and the Colorado Plateau in Arizona and New Mexico (Fig. 2) and violations are expected in Utah and Colorado when these states report. Thorium enrichment zones, such as veins and placer deposits, are expected to produce only scattered, local Ra-228 problems, due to its limited transport in ground water. These areas would be extremely difficult to locate under the present regulations. However aquifers with much lower but disseminated thorium and uranium (such as granites, tuffaceous rocks, and immature sandstones) are more likely to have higher background radioactivity and wider occurrences of both Ra-228 and Ra-226 in ground water.

## Occurrence of Ra-224

Data on Ra-224 activities in ground water are scarce. However, it appears that the activity of Ra-224 is equal to or as much as twice the Ra-228 activity and therefore could be as high as 30-40 pCi/l. This Ra-224 activity is unsupported; activities of its parent, Th-228, are usually less than 0.01 pCi/l. Thus it enters ground water by alpha recoil during decay of Th-228 adsorbed on the surface of aquifer solids. The radiotoxicity of Ra-224 and its daughters is small because of their extremely short half-lives.

## RADIUM CONCLUSIONS

From the state compliance data and other studies, much more is now known about the occurrence of radium isotopes in public drinking water supplies, and this information should be incorporated into the revised regulations.

1) Surface water has very low radium activities; the monitoring interval after initial validation should be significantly lengthened, or perhaps omitted, for surface-water systems.

2) Fewer samples may be needed to determine the average annual activities, particularly for single-well systems; the monitoring interval could be lengthened for unmodified systems.

3) Monitoring requirements for Ra-228 should be decoupled from Ra-226. Instead, separate guidelines for the occurrence of radium isotopes are needed.

4) Regional Ra-226 problems are fairly well known. The few additional occurrences of high Ra-226 activity will be difficult to find without analysis of every system. As important, however, are those areas which have low Ra-226. All values should be compiled regionally or nationally, to document the Ra-226 distribution for each geological province, with the goal to classify areas with a high degree of certainty as producing low Ra-226 ground water. The revised regulations should include separate, less stringent and less costly monitoring requirements for such regions. This approach would shift monitoring efforts toward known or uncertain areas of high Ra-226 and provide more data on the actual distribution of high Ra-226 activities, which will allow for a better risk assessment.

5) The occurrence of Ra-228 is not well known. It has been shown that, using the present screening procedures, 10 to 50 percent of the violations for total radium are being missed. More extensive measurements of Ra-228 would be difficult because of the problems with the approved analytical method. An alternative approach would be to develop a conceptual, predictive model for Ra-228 occurrence, based on geochemical principles, to identify specific types of aquifers which are likely to have Ra-228 problems. Once verified, this model should be the basis for developing regional guidelines for monitoring in areas more likely to have high Ra-228. This same approach can be used for refining and interpreting occurrence data for uranium and radon as part of the regulatory process of developing standards for these isotopes.

6) Finally, because aqueous radiochemistry is a complex, technical field, EPA should provide State water-supply personnel with background and explanatory guides in laymen's language, which will assist them in understanding radiological problems and in the implementation of the regulations.

# OCCURRENCES OF URANIUM ISOTOPES IN PUBLIC DRINKING WATER

## INTRODUCTION

Uranium has three natural isotopes with long half-lives ($t_{1/2}$) that permit transport into potable water supplies. These isotopes are uranium-238 (99.27% natural abundance), $t_{1/2}$ = 4.7 x $10^9$ years, uranium-235 (0.72% natural abundance) $t_{1/2}$ = 7.04 x $10^8$ years, and uranium-234 (.006% natural abundance) $t_{1/2}$ = 2.54 x $10^5$ years. All of these isotopes emit alpha radiation and produce a long decay series of daughters. The group of uranium isotopes are found in the Earth's crust with an abundance of 4 x $10^{-4}$% (Hu73) and are found in rocks and minerals such as granite, metamorphic rocks, lignites, monazite sand, and phosphate deposits as well as in uranium minerals such as uraninite, carnotite and pitchblend (Ca80). It is a trace element in coal, peat, and asphalt and is present in some phosphate fertilizers at a level of about 100 micrograms/g or 67 pCi/g. Despite its widespread abundance it has not been shown to be an essential element for man (Hu73). There is no standard for uranium in water supplies as a radioactive element since, until recently, it has been considered by the NRC to be a toxic heavy metal with the standard for ingestion relating to its chemical toxicity (3 x $10^4$ pCi/l), (10CFR 20,601, Appendix B). However, some concentration measurements in potable water have been done in association with gross alpha measurements for the radium drinking water standard. The uranium activity measured was to be subtracted from the gross alpha activity measurement to show compliance with the gross alpha standard (Dr81). A recent analysis of uranium in water supplies was conducted by Oak Ridge National Laboratory (Dr81) using 89,944 measurements of uranium surface, ground, and domestic waters primarily obtained from the National Uranium Resource Evaluation (NURE) program. The results of this study are reviewed in this report.

## GEOCHEMISTRY OF URANIUM

Although there are geological processes which enrich uranium in certain rock formations, it occurs as a common trace element in most rock types. Because of the insolubility of $U^{4+}$, uranium must be oxidized in order to be transported in ground water. The greater solubility of $U^{6+}$ is due in part to its tendency to form uranyl di-and tri-carbonate anions. Thus, uranium solubility is a function of not only the redox potential of water but also of the pH and the partial pressure of $CO_2$ in the system. In comparison to radium, the stability of the uranyl carbonate complexes and their long half-life allow for uranium to be transported long distances under oxidizing conditions. Uranium is removed from solution by sorption or reducinig barriers, a process which has been well described in the sandstone-type uranium deposits in the western United States (Ga77).

There have been many studies of the isotopic composition of uranium in natural waters which have shown that most contain more activity from U-234 than from U-238. The U-234/U-238 activity ratio can be as high as 28, but usually ranges between 1 and 3 (Ch71, Gi82). The higher activity of U-234 in water is due to its selective mobilization by alpha recoil. The natural abundances of isotopes and the half-lives gives 0.33, 0.015 and 0.33 pCi/µg of natural uranium for U-238, U-235 and U-234, respectively, or 0.68 pCi/µg total. Thus, isotopic enrichment can cause changes in the specific activity of the total sample of uranium. Total depletion of U-234 from the sample and replacement by an equal activity of U-238 will result in no net change of total activity; however, the total mass of uranium would almost double. The human dosimetry will also be changed since the alpha energies are not the same. Methods which depend on the mass of uranium will not predict the correct activity for samples with variable uranium isotope enrichment.

## OCCURRENCE OF URANIUM IN GROUND AND SURFACE WATER

Uranium concentration in water depends on factors such as the uranium concentration in host aquifer rock, presence of oxygen and complexing agents, chemicals, in the aquifer, chemical reactions with ions in solution and the nature of the contact between the uranium minerals and the water. These factors vary (Sc62) with regions of the United States due to rainfall, geology, and ground water flow patterns, and to anthropogenic factors such as use rate of ground water and surface water. Thus, one would expect large variations of uranium content from state-to-state. The geological provinces of the coterminous United States derived from generalizations of rock types and hydrological flow systems are shown in Figure 1 and Table 1. These zones can be compared to the uranium concentrations averaged by state from the (NURE) measurements shown in Figures 3, 4, 5. These averages are surprisingly similar, showing differences of only factors of 4 higher concentrations in ground water than in surface water, with many states identical for ground and surface water. The average values for each province is given in Table 4 with average values ranging from .02 in province 2, the Appalachian Mountains, up to 2.3 in province 8, the Colorado Plateau. By grouping low, medium, and high averages, one sees the four major zones of similar concentration; Zone 1, the Appalachian Mountains and New England; Zone 2, Appalachian and Interior Plateau, and Coastal Plain; Zone 3, the Glaciated Central Platform, Western Central Platform, Rocky Mountain System, and Colorado Plateau; Zone 4, the Basin and Range and the Columbia Plateau and Pacific Mountain System.

The provinces chosen by Beddinger (Be81) may be compared with those chosen in 1962 by Scott and Barker (Sc62). These provinces are shown in Figure 1 and compared by region in Table 1. Concentrations of uranium are given in Table 4 for provincial schemes. The data of Scott and Barker comprise 561 samples collected in the coterminous United States from 1954 to 1958 and are expressed in μg/L (thus they represent U-238 only).

Table 5 shows population versus uranium concentration for drinking water sources with more than 10,000 people (Dr81). The levels are given up to greater than 100,000 population. Due to limitations in the source information (Dr81), no information is available for cities of populations less than 10,000, showing a need for more information on small systems.

## RELATIVE SOURCE CONTRIBUTION OF URANIUM

The dietary intake of uranium in United States food is variously reported from 0.87 to 0.94 picocuries per day (Ha73) to 0.2 to 0.9 pCi/day (UN77) with an average of 0.4 pCi/day. The comparison with drinking water of average concentration of 2 pCi/L and 2 liter per day consumption gives 4 pCi/day of water derived uranium which is five to ten times greater than the food derived uranium. Air contributions of uranium are much smaller than the food and water contributions.

## URANIUM CONCLUSIONS

1. The data shows that elevated levels of uranium found are found in surface water as well as in ground water.

2. Highest average values of uranium concentration are found in decreasing order in the following provinces: Colorado Plateau, Western Central Plateau, Rocky Mountain System, Basin and Range and Pacific Mountain System. The highest state is South Dakota. Modeling these variations would be very helpful for regional standards.

3. High uranium concentrations in the East are widely separated, most values in the East are low.

4. Isotopic estimates are needed for $^{238}U$, $^{234}U$ since they are found in disequilibrium in water, regional variations should be modeled.

5. More analyses are needed for low population systems.

## OCCURRENCE OF RADON ISOTOPES IN PUBLIC DRINKING WATER

### INTRODUCTION

There are two isotopes of radon, with half-lives long enough to be considered as drinking water radionuclides. The first is Rn-222 which is the daughter of Ra-226, called "radon", and has a half-life of 3.84 days. The second Rn-220 which is the daughter of Ra-224, was historically called "thoron", and has a half-life of 56 sec. The time delay from production to consumption of water of a few hours to a few days for water allows many decay half-lives for the Rn-220, and it is not observed in water supplies. Rn-222 henceforth simply radon, is transported by the water, can lead to public exposures by being ingested and exposing the digestive system and by becoming airborne and exposing the lungs. When water is used for cleaning, dish-washing, bathing, or clothes washing, radon escapes from the water into building air where it decays into alpha emitting daughters. The resulting radon daughters are charged and will frequently attach to aerosol particles in the air. The dust, cigarette smoke, or aerosol particles will then be inhaled and may become attached to the interior of the lung, bringing the alpha particle emitting radon daughters into close association with the cell lining of the respiratory system (Ar75).

There is no federal standard for radon in water, although studies on ingestion doses (Hu65) (As79) and inhalation doses (Pr81), (He83a), and (He79) have been done. There have been standards for radon in mine air, and for radon from soil gas in buildings placed on mine tailings in the United States and Canada (Us79, At77). Sweden has standards for radon from soil gas in areas of alum shale and granites (Ak81). Some of these standards are given in Table 6. In the past, radon and its daughters have been excluded from the drinking water standards, and considered only to be an interference in the radium measurements.

Information about levels of radon have been obtained in state studies and by a federal study done by the U.S.E.P.A. Environmental Radiation Facility in Montgomery, Alabama and by the University of Texas at Houston.

## RADON - RADIUM GEOCHEMISTRY

Radon is a water soluble inert gas and its occurrence is controlled by physical variables such as pressure, temperature, emissivity of radon from rocks, as well as by time, and by the geochemistry of its parent Ra-226. High activity of radon is associated with granitic rocks (St81), uranium minerals (Ta78), such as uraninite, carnotite and with tailings from phosphate fertilizer processing (Us79) and uranium mines.

## TRANSPORT OF RADON IN WATER FROM ROCKS

As discussed in the geochemistry section, the occurrence of radon in water is controlled by chemical concentration of radium in the host soil on rock and by emissivity of radon into the water. The physical condition of the rock matrix appears to play a greater role in radon production than does the concentration of parent radium. Several investigators (An72), (Ra83), (Ta64), and Ta80) examine the mechanisms influencing the release of radon from rock grains and the transport of radon through an aquifer. Experimental and theoretical considerations indicate that diffusion along microcrystalline imperfections dominates the release of radon into the surrounding interstitial waters. The movement of radon in water is governed by water transport rather than diffusion in most cases, i.e., cases in which the percolation velocity is greater than $10^{-5}$ cm/sec.

The variation of radon concentration with rock type for well water in Maine is illustrated in Table 7. This variation of a factor of 20 illustrates variation in rock types in Maine. Public utility water measured at the wells, which is also presented in the Maine list, is a factor of 5 lower than the state average. This is due to the higher capacity of the gravel aquifers used for utility water supplies. These gravel aquifers allow more water to mix with the same amount of radon, leading to lower concentrations of radon.

## OCCURRENCE OF RADON IN PUBLIC WATER SUPPLIES

Concentrations of radon in various water sources conform to the log normal distribution. Table 8 shows the results of a blind sampling of public water supplies in the central United States. A reanalysis of other published data (Table 9) shows a similar trend. Some sources appear to be samples from a single log normal distribution, others from two, or perhaps three distributions, as indicated by the sharp breaks or bends in the plots. For this reason geometrical averages are used for the samples shown below.

The occurrence of radon in public ground water supplies in the United States is shown in Figure 7 (taken at the tap) and Table 9 (mainly utility samples). Radon activities are thousands of times higher than uranium or radium, probably due to absorbtion of the radium and uranium by the host rock (Ki82). The results of these geometric means show highest values in mountain states especially in the Appalachians with the highest states Rhode Island, Maine, New Hampshire, Vermont, Massachusetts, Pennsylvania, and Virginia. California is the highest western state. The high radon values associate with the granitic areas in the Appalachian Highlands Piedmont Provinces (Figure 1). Midwestern and coastal plane values are lower and mountain states in the west are higher. Figure 8 shows the private ground water supplies (mainly individual samples) for the United States. These results of geometrical averages show the private supplies are higher by a factor of 3-20 times the public ground water samples. This factor results from the use of low capacity wells for private supplies while public supplies use high capacity sand or gravel wells. The higher states in the private well list are Rhode Island, Florida, Maine, South Dakota, Montana and Georgia. Larger numbers of samples would be desirable to strengthen these conclusions. Public surface supplies have radon concentrations less than 100 pCi/l. Table 10 shows a breakdown of the radon concentrations in water by state and by population of the town using the well. Highest radon concentrations are found in the less than 100 category (see Maine and US for examples). The United States geometric population average is 187 pCi/l.

## MAJOR SOURCES OF INDOOR RADON

Radon produced from radium in the surficial soil and rock (Ak81) is released into houses from water, soil gas, fuel gas and construction materials and outdoor air. Both water and soil gas can be transported into buildings through cracks, drain holes, as well as water and fuel gas supply pipes (Sc82). The radon in the ground water is released as it is mixed with air in such indoor uses as cleaning, bathing, dish and clothes washing and toilet flushing (He83a) (Pa79). Soil gas will mix into building air and then diffuse throughout the house. Radon from fuel gas enters building air from unvented heaters or stoves. Thus, the radon concentration in air will depend on the sum of all radon sources (Ge78), the volume of the building, and the ventilation rates of the building (Fl80) (Ne81). The average value for radon in house air due to all these sources has been estimated at 0.3 - 2.2 pCi/l in normal regions, 1.1 - 1.67 in anomalous regions of the U.S. (Br81).

### The Soil

Radon diffuses from the soil through cracks in foundations, unventilated crawl spaces, basement drains, and other pathways into the living space. Direct outgassing from the soil is the dominant source of indoor radon in most cases contributing .03 - 1.5 pCi/l in normal regions, 0.3 - 15 pCi/l in anomalous regions.

If most radon enters structures through the basement or foundation, radon concentrations would tend to decline markedly with story above ground, as shown in Table 11. The limitations of building materials and ground waters as sources of radon (see below) coupled with the tendency toward single distributions in given areas combine to suggest that the soil is generally the largest source.

Ground Water

Ground waters containing radon can add substantially to the amount of radon in the air of a dwelling. Much of the dissolved radon can escape when water is used for various domestic purposes inside a dwelling. The amount of radon in indoor air due to the use of water depends a great deal on architectural and life-style related variables. The most sensitive dwellings will be small, relatively tight structures in which large amounts of water are routinely used in household appliances. A model for the average increment to the indoor atmosphere can be expressed as:

$$C_a = \frac{C_w}{24RV} \sum e_i w_i$$

where $C_a$ and $C_w$ are the concentrations (pCi/l) of radon in the air and water, respectively, R is the air change rate ($hr^{-1}$), V is the dwelling volume, $W_i$ represents the average amount of water (l) used daily in the ith domestic application, and $e_i$ denotes the transfer efficiency, or the fraction of radon released to the air for the ith application.

A number of investigators have made semi-empirical determinations of $C_a/C_w$; most are on the general order of $10^{-4}$ pCi/l in the air per pCi/l in the water. Table 12 shows a number of such estimates and the underlying assumptions used in their derivation. Using Table 12 the range of values for radon in air from water in the U.S. ranges from 0.2 pCi/l average for Rhode Island to as low as $1.2 \times 10^{-3}$ pCi/l average for Tennessee.

The relative importance of water as a source of indoor radon will of course depend upon the amount of radon in the water and the magnitude of other sources. Upper limit calculations on one recent data set (Table 12)(Pr83a) show water as the source of up to 35% of the net (indoor minus outdoor) radon observed in a set of 81 bedrooms in single family houses in the State of Maine. The highest radon levels were seen in the basement, which suggests that the soil is a major source of indoor radon. The long-term average levels noted in the bathroom were also higher than those noted in the other living areas, however. Both the integrated indoor air radon concentrations and the concentrations of radon in water were distributed log-normally, and a ratio of 4 pCi/l (bedroom air) per $10^5$ pCi/l (water) was found by regression. The application of this factor to the geometric mean of the water distribution led to a predicted increment of 0.23 pCi/l in the air versus a net bedroom concentration of 0.66 pCi/l. This is an upper limit estimate, based on the assumption that the magnitudes of other sources of radon are independent of the amount of radon in the water. In fact, the concentration of radon in the basements was found to be correlated with the concentrations of radon in water at the $p = 0.01$ level.

## Construction Materials

A number of recent publications have addressed the problem of elevated indoor radon concentrations arising from the use of building materials containing radium-226 (Br81). A portion of the radon arising from construction materials is able to diffuse into the living area, where the ultimate concentration increment will depend on the volume of the dwelling and the ventilation rate ranging from .003 pCi/l to 0.3 pCi/l in the U.S. (Br81). Perhaps the best publicized case involving construction materials occurred in Grand Junction, Colorado, where uranium mill tailings were once frequently used as fill materials around foundations. Over 5000 buildings were associated with tailings material to some extent, and 3000 of those buildings were built on top of a layer of tailings. When radon levels in some of the dwellings were found to be markedly elevated a general survey was conducted, and eventually a remedial action program was implemented for those structures exceeding national standards. (See Table 6). A similar situation arose in the phosphate mining area of Florida. Dwellings were built on mining lands reclaimed at least in part with phosphate rock residues. Radon concentrations well over 10 pCi/l have been noted in buildings containing materials such as gypsum wall board derived from phosphate residues or cinderblocks made of fly ash or blast furnace slag.(US79).

## Fuel Gas

Radioactivity in natural gas was noted as early as 1904 (Sa18), but it was not until the 1970's that the potential contribution to indoor radon concentrations was seriously investigated. Interest in the health implications of naturally occurring radon in gas deposits was stimulated by investigations of the practicality of stimulating natural gas yields by detonating a nuclear warhead in the appropriate rock formation (Bu66). Estimates of dose increments caused by the combustion of natural gas in the home were made by Barton (Ba73), Johnson (Jo73), and Gesell (Ge74). Similar calculations were made for liquified petroleum gas (LPG), which, because of boiling point considerations, contains a higher concentration of radon than the natural gas from which it is made.

The increment to the indoor environment depends on the amount of gas of LPG burned in unvented ranges or heaters, the size and infiltration characteristics of the dwelling, and of course, the concentration of radon in the fuel. The contribution from this source is usually quite small (0.15 pCi/l) due to the low use of unvented heaters (Ge77).

## OCCURRENCE OF RADON IN INDOOR AIR

There are a number of situations in which indoor radon levels are especially elevated. These situations occur when the structure contains a stronger than usual source of radon, or when the structure has especially low ventilation and infiltration rates, or both. Rising heating and air conditioning costs in the last several years have encouraged people to reduce air infiltration rates.

A number of trends can be discerned in the recent literature. Within a given locale, indoor radon concentrations tend to be distributed log-normally, and sample means vary markedly from area to area (Ge83). It is becoming increasingly apparent that local geological factors play a major, if not dominant role in determining the distribution of indoor radon concentrations in a given area.

### Concentrations of Radon In The Indoor Environment

One of the most extensive studies of radon in dwellings on record is a recent survey of 12,000 Swedish homes (Hi81). All the dwellings involved were ones in which elevated radon levels were expected. The results as shown in Table 13 are reported in working levels, and the presumed associated radon concentrations, based on an equilibrium ratio of 0.5, are added in parentheses.

The measurements summarized above are not meant to be representative and are probably considerably higher than the true area average. Nevertheless, the number of dwellings involved and the high values observed combine to demonstrate the extent of the radon problem in certain areas. Nearly half of the 12,000 dwellings were found to exceed current U.S. occupational standard for uranium mines (adjusted for constant occupancy vs. a 40 hour week).

Another extensive survey involved nearly 10,000 randomly selected houses in 14 Canadian cities (Mc80). Single grab samples were obtained from basements or the ground floor during the summer months. In general, data from a given city were better fit by the log-normal distribution than by the normal distribution. The geometric means ranged from 0.14 pCi/l in Vancouver to 0.88 pCi/l in St. Lawrence, Newfoundland. The geometric standard deviations ranged from 2.78 to 6.77.

Table 11 summarizes a number of surveys conducted in the United States in areas not known or suspected to involve anomalies due to mill tailings or unusual mineralization. The data presented are either the average of a number of grab samples taken within a single dwelling, or were developed by long-term measuring devices. The equilibrium fraction (f) of radon daughters is given where available.

## RADON CONCLUSIONS

1. Radon concentrations in water are highest in ground water especially in granite areas. Radon concentrations in surface water are very low.

2. Higher concentration occurs in small systems. Domestic supplies have higher concentrations of radon in water than public wells. Utility systems are lower by a factor of 10 than private wells.

3. The highest average radon concentrations in water are found in the provinces in decreasing order: New England, Appalachian Highlands-Piedmont, Pacific Mountain System, Rocky Mountain System.

4. Ventilation affects radon concentration in air with an approximate value of 1. x $10^{-4}$ for the ratio of radon concentration in air to radon concentration in water for a house with one air change per hour. Soil gas radon contributes a sizeable portion of the total radon in air.

5. Additional measurements of radon in systems serving less than a thousand users are required in order to better quantify exposures to the group that potentially represents the highest population dose. Because the number of such systems is quite large (~37,000), these measurements should be obtained from a representative sampling program guided by geological models.

## CALCULATION OF WATER UTILITY RISKS FROM RADIONUCLIDES IN WATER BASED ON NATIONAL OCCURRENCE DATA

Each nuclide present in drinking water will present a risk to the utility users of the water which is related linearly to the occurrence, concentration, population exposed, and the individual risk rate (Ma83). The case of radium in drinking water data allows a direct calculation. The average concentration of radium in drinking water from utilities is 1.6 pCi/l (Ho83), and the population consuming this water is 70 million people which is the half of the U.S. population which uses ground water provided by utilities. Since the individual risk rate is hypothesized to be by linear dose response 24.5 x $10^{-6}$ excess cancers/lifetime person pCi/l (using Ma83), we can calculate

$$1.6 \text{ pCi/l} \times 24.4 \times 10^{-6} \frac{\text{excess cancers}}{\text{lifetime person}} \; \frac{1}{\text{pCi/l}} \times 70 \times 10^{6} \text{ people}$$

$$\sim \frac{\text{2688 people}}{\text{lifetime in U.S.}} .$$

Even the elimination of bone sarcomas at low concentration will leave the sinus carcinomas which are half of this number (Ma83). An additional 30 x $10^6$ people are exposed at less than .5 pCi/l surface water provided by utilities. The distribution of occurrence of radium concentrations permits estimates of the number of fatal cancers averted when the standard is placed at a particular concentration. Since the standard is at 5 pCi/l, it seems reasonable to estimate the fraction of cancers averted by the standard. The average for the supplies of greater than 5 pCi/l is 8 pCi/l. This concentration is multiplied by an estimate of the population which uses those supplies obtained from rounding the average ground water utility population from the U.S.E.P.A. Summary.

$$8\ \text{pCi/l} \times \frac{1000\ \text{persons}}{\text{supply}} \times 500 \times \frac{24.0 \times 10-5}{1\ \text{pCi/l}} \qquad \frac{\text{excess cancers}}{\text{person lifetime}}$$

$$\sim 96\ \frac{\text{excess cancers}}{\text{lifetime}}$$

Population risk can also be done for uranium in water by using a similar calculation. For uranium the population weighted average radioactivity concentration is 0.8 pCi/l for the whole U.S. population (Dr81). This permits this result for the risk using individual risk factors from (Ma83), (Wr83) and (Co83).

$$[0.8\ \text{pCi/l}]\ \frac{[100\ \text{mrem/yr}]}{10\ \text{pCi/l}} \times 220 \times 10^{6}\ \text{people exposed}$$

$$\frac{\times\ 34 \times 10-6}{100\ \text{mrem/yr}}$$

$$= \frac{598\ \text{persons}}{\text{lifetime in U.S.}}$$

Since there is no drinking water standard for uranium, at this time, we must use the fraction of people at each occurrence level to estimate the number of cases avoided by a standard for water utilities.

Using values from Drury *et*. *al*. a water utility users risk estimate table can be formed (Dr81) (See Table 15.)

The population risk for radon in utility water supplies can be calculated using radon concentration in utility water, exchange rate from water into air, cancer rate per working level month per million population and the exposed population in millions. From the work done by the committee on the Health Effects of Radon in drinking water (Cr83), we can obtain the individual risk factor which includes the exchange rate for radon from water into air and the cancer rate pr working level month per million population exposed to air for a lifetime of 20 years. This factor is $3 \times 10^{-7}$ lung cancers per pCi/l radon in water. This factor is increased to $4 \times 10^{-7}$ cancers when stomach and whole body cancers are included (Cr83). The radon in water concentration data for the whole United States are geologically controlled and are generally a mixture of low values around 100-200 pCi/l and high values of 10,000-1,000,000 pCi/l. This extreme range of values leads to arithmetic averages which are strongly influenced by the highest few points. The geometric mean of these data will average the numbers with less weight for the high values. This geometric mean will be lower than the arithmetic mean. We have decided to calculate the risk with both of these means.

Using data from the geometrical and arithmetic means of radon concentrations for utilities of different sizes, and the geometrical and arithmetic risk factor, we can calculate the population at risk for the utilities serving less than 100 population, 100-1000, population, 1000-5000 population, 5000-10,000 population, 10,000-100,000 population, and 100,000 and above population. Lifetime risk is shown for both geometric averages and arithmetic averages in the right column of Table 16.

## OTHER NATURAL RADIOISOTOPES OF POSSIBLE CONCERN

The natural radioisotopes discussed above (U, Ra-228, Ra-226, and Rn-222) are of greatest concern because of their long half-lives and the health risks associated with the activities that can be present in public drinking water. There are two classes of other natural radioisotopes that may be of possible concern: (1) relatively long-lived isotopes whose activity is derived from the aquifer, termed unsupported (Th-232, Th-230, Pb-210, Po-210) and (2) very short-lived isotopes which "grow in" once the ground water is pumped from the aquifer and thus are supported (primarily Rn-222 daughters). However, very few data are available on the activities of these isotopes in drinking water, primarily due to their low solubility and/or the difficulty of measuring isotopes of short half-lives.

Table 17 shows the typical ranges of activities of the longer-lived radionuclides in ground water. Activities in surface waters will be extremely low (except perhaps in hot springs) due to rapid sorption onto suspended and bottom sediments. The highest known activity of Th-232 and Th-230 in U.S. drinking water is from a well in California that also contained large amounts of dissolved organic matter which probably complexed with thorium. Most other values are below 0.1 pCi/l. Th-230 would be expected to be slightly higher than Th-232 due to generation by U-234 in solution and by alpha recoil. Likewise, detectable activities of Pb-210 and Po-210 would be expected because of the relatively large amount of Rn-222 present in many ground waters. The insolubility of these isotopes and their short-lived precursors in the aquifer is demonstrated by the fact that more than 99.9 percent of the activity generated by Rn-222 decay in ground water is removed within the two hours necessary for equilibrium to be established between Rn-222 and Pb-210. The only known anonymously high Po-210 value in drinking water is the surprisingly large activity in Louisiana for which the source has not been determined. In general, these longer-lived isotopes are not expected to occur in activities greater than 1.0 pCi/l.

The second class of radioisotopes of possible concern are Rn-222 daughters which reach equilibrium with Rn-222 within two hours. In untreated ground water systems, removal by adsorption would not be as rapid as in the aquifer due to the small surface area of the distribution system. Exposure from consumption of these supported, extremely short-lived daughters should be evaluated. Rn-220 and its daughters do not pose a similar problem primarily because its 54.5-second half-life is too short to allow diffusion out of the aquifer materials and the initial activities are much lower.

would not be as rapid as in the aquifer due to the small surface area of the distribution system. Exposure from consumption of these supported, extremely short-lived daughters should be evaluated. Rn-220 and its daughters do not pose a similar problem primarily because its 54.5-second half-life is too short to allow diffusion out of the aquifer materials and the initial activities are much lower.

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TABLE 1--Summary of potential host rocks, geologic framework, and nature of ground water flow systems in the Provinces of the coterminous United States (Fig. 1)

| PROVINCE | GEOLOGICAL FRAMEWORK | NATURE OF GROUND WATER FLOW SYSTEMS |
|---|---|---|
| 1. New England Adirondack Mountains | New England--complexly faulted metamorphic and metasedimentary rocks intruded by large masses of granite. Adirondacks--mountains composed of marble and schist intruded by granites, anorthosite, and gabbro. | Flow and head in bedrock of low permeability and overlying glacial aquifers greatly influenced by local topography and surface-water features. |
| 2. Appalachian Highlands-Piedmont | Appalachian Highlands--mountain belt of granites and metamorphics thrust westward over Paleozic rocks. Piedmont--non-mountianous belt of highly complex metamorphic rocks with abudant granites. | Flow and head in metamorphic and granite bedrock of low permeability, largely controlled by topography and surface water features; folded limestone locally cavernous and highly permeable at shallow depths supporting large springs; sandstone aquifers of moderate extent and permeability; flow systems generally related to local recharge in interstream areas and discharge to surface water features. |
| 3. Appalachian and Interior Plateaus | Appalachian and Interior Plateaus consist of gently dipping, gently folded, sandstones, shales, carbonates, and evaporites. In southern Missouri exposing old crystalline rocks. | Regional flow in low to moderately permeable sandstones and carbonates; carbonates locally of high permeability at shallow depth due to fractures and solution channels support large springs. |

TABLE 1-- Summary of potential host rocks, geologic framework, and nature of ground water flow systems in the Provinces of the coterminous United States (Fig. 1)--Continuous

| PROVINCE | GEOLOGICAL FRAMEWORK | NATURE OF GROUND WATER FLOW SYSTEMS |
|---|---|---|
| 4. Coastal Plain | Seward dipping thickening wedge of sand, sandstones and shales with some evaporites and limestones; underlain by a basement of metamorphic rocks. | Regional flow in sand and limestone aquifers with intervening clay confining layers; predominant flow direction seaward; discharge upward through confining layers and to streams. |
| 5. Glaciated Central Platform | Igneous and metamorphic rocks on the northwest overlain by sandstones, carbonates, shales, and evaporites; deep basin deposits in Michigan and Illinois. | Regional flow in sandstone and carbonate aquifers; highly mineralized water at depth in basins; glacial aquifers locally overlie bedrock. |
| 6. Western Central Platform | Horizontal to gently dipping sandstones; deep sedimentary basins and structural high. Capped with sands and gravels. | Regional flow in layered sandstone and carbonate aquifers; thick confining beds of shale; deep basins contain highly saline water. Extensive fluvial deposit aquifers Nebraska south into Texas and glacial aquifers in North Dakota and South Dakota overlie sandstones. |

TABLE 1--Summary of potential host rocks, geologic framework, and nature of ground water flow systems in the Provinces of the coterminous United States (Fig. 1)--Continuous

| PROVINCE | GEOLOGICAL FRAMEWORK | NATURE OF GROUND WATER FLOW SYSTEMS |
|---|---|---|
| 7. Rocky Mountain System | Igneous and metamorphic folded core rocks of Rocky Mountains and intermontane basins of shales, carbonates, evaporites, and sandstones. Intrusive and volcanic rocks. | Regional flow in layered sandstone and carbonate aquifers with shale confining beds in intermontane basins; local recharge and discharge controlled by topography and surface water features in fractured igneous and metamorphic rocks. |
| 8. Colorado Plateaus | Flat-lying to gently warped layers of sandstones, shales, limestones and evaporites with volcanic rocks. | Regional flow in layered sedimentary rocks; chief aquifers are sandstones and carbonates; discharge to major streams; highly saline water at depth in deep basins and in association with salt beds. |
| 9. Basin and Range | Elongate blocky mountains of faulted rock complexes; deep alluvium-filled intermontane basins; intrusive igneous stocks and plugs; extrusive ash-flow tuffs, rhyolites, and basalts. | Flow within closed basins; inter-basin flow between closed topographic basins through permeable bedrock; interbasin flow in alluvial channels between basins with integrated surface drainage; deep regional flow systems in carbonate and volcanic rocks. |

TABLE 1--Summary of potential host rocks, geologic framework, and nature of ground water flow systems in the Provinces of the coterminous United States (Fig. 1)--Continuous

| PROVINCE | GEOLOGICAL FRAMEWORK | NATURE OF GROUND WATER FLOW SYSTEMS |
|---|---|---|
| 10. Columbia Plateaus | Regional shallow structural basin of basaltic lava flows; locally faulted and folded; mountain range on the west consisting of elongate chain of andesitic volcanic cones. | Basaltic lava flows range from highly permeable to dense nearly impermeable creating regional aquifers with perched aquifers separated by confining beds. The ground water principally discharges to the major streams; local discharge to a few closed basins. |
| 11. Pacific Mountain System | Consists of several complex elements; Large uplifted and tilted blocks of granite with inliers of metasediments; folded and faulted folded and faulted sedimentary rocks; deep elongate troughs filled with fluvial sediments. | Regional flow in deep intermontane sedimentary basins; igneous and metamorphic rocks of low permeability support shallow local flow systems related to topography and surface drainage. |

TABLE 2. Summary of Ra-228 and Ra-226 Distribution in Ground Water by Aquifer Type for the Atlantic Coastal Plain and Piedmont Provinces.

| Aquifer Type | Number of Values | Ra-228 | | Ra-226 | |
|---|---|---|---|---|---|
| | | Geometric Mean (pCi/l) | Range (pCi/l) | Geometric Mean (pCi/l) | Range (pCi/l) |
| Igneous Rocks (acidic) | 42 | 1.39 | 0.0-22.6 | 1.80 | 0.0-15.9 |
| Metamorphic Rocks | 75 | 0.33 | 0.0- 3.9 | 0.37 | 0.0- 7.4 |
| Sand | 143 | 1.05 | 0.0-17.6 | 1.36 | 0.0-25.9 |
| Arkose | 92 | 2.16 | 0.0-13.5 | 2.19 | 0.0-23.0 |
| Quartzose | 50 | 0.27 | 0.0-17.6 | 0.55 | 0.0-25.9 |
| Limestone | 16 | 0.06 | 0.0- 0.2 | 0.12 | 0.0- 0.3 |

TABLE 3. Ranking of all Structural Levels used Showing Groupings of Similarity in Ra-228 Distribution. Ra-226 Values are given for comparison but are not ranked.

| Grouping* | | | | Mean Ra-228 (pCi/l) | No. | Structural Level | Mean Ra-226 (pCi/l) |
|---|---|---|---|---|---|---|---|
| | | A | | 3.03 | 2 | Igneous, acidic, composite Th, | 0.75 |
| | | A | | | | syenite | |
| | | A | | 2.49 | 46 | Sand, unconsolidated, arkosic, | 2.03 |
| | | A | | | | high-Th source | |
| | | A | | | | | |
| B | | A | | 2.14 | 43 | Sand, unconsolidated, arkosic, | 2.73 |
| B | | A | | | | medium-Th source | |
| B | | A | | | | | |
| B | | A | C | 1.59 | 35 | Igneous, acidic, composite Th, | 2.31 |
| B | | A | C | | | granite | |
| B | D | A | C | 0.85 | 3 | Igneous, acidic, composite Th, | 0.99 |
| B | D | | C | | | diorite | |
| B | D | E | C | 0.52 | 31 | Sand, unconsolidated, quartzose, | 1.72 |
| | D | E | C | | | medium-Th source | |
| | D | E | C | | | | |
| F | D | E | C | 0.39 | 37 | Metamorphic, high-grade, | 0.32 |
| F | D | E | | | | nonspecific Th | |
| F | D | E | | 0.34 | 10 | Metamorphic, low-grade, | 1.41 |
| F | D | E | | | | nonspecific Th | |
| F | D | E | | 0.28 | 3 | Sand, unconsolidated, arkosic, | 0.32 |
| F | D | E | | | | low-Th source | |
| F | D | E | | | | | |
| F | D | E | | 0.28 | 13 | Metamorphic, medium-grade, | 0.24 |
| F | D | E | | | | nonspecific Th | |
| F | D | E | | | | | |
| F | D | E | G | 0.24 | 15 | Metamorphic, high-grade, | 0.24 |
| F | D | E | G | | | specific Th, monazite | |
| F | | E | G | | | | |
| F | | E | G | 0.12 | 2 | Igneous, acidic, refractory Th, | 0.14 |
| F | | E | G | | | monazite | |
| F | | | G | | | | |
| F | | | G | 0.09 | 19 | Sand, unconsolidated, quartzose, | 0.09 |
| F | | | G | | | low-Th source | |
| | | | G | | | | |
| | | | G | 0.06 | 16 | Chemical precipitates, limestone | 0.12 |

*A>B>C>D>E ($p<0.05$).

TABLE 4

Uranium Concentration in the Provinces

| | Beddinger's Uranium concentrations (pCi/l) | | Scott's Uranium concentrations (pCi/l) |
|---|---|---|---|
| Beddinger Provinces | Arithmetic Mean | Standard Deviation | Arithmetic Mean |
| 1. New England--Adirondack Mts. | 0.46 | 0.03 | ] II .34 |
| 2. Appalachian Highlands Piedmont | 0.020 | 0.013 | |
| 3. Appalachian Interior Plateaus | 0.137 | 0.27 | III .34 |
| 4. Coastal Plain | 0.108 | 0.19 | I .14 |
| 5. Glaciated Central Platform Aquifers | 1.04 | 2.17 | V .34<br>VI .71 |
| 6. Western Central Platform | 2.1 | 2.2 | VI 1.5 |
| 7. Rocky Mountain System | 1.99 | 1.27 | VII-VIII 1.15 |
| 8. Colorado Plateaus | 2.31 | 1.4 | IX .54 |
| 9. Basin and Range | 2.15 | 1.53 | VIII 1.15 |
| 10. Columbia Plateaus | 0.52 | 0.389 | ] X 0.14 |
| 11. Pacific Mountain System | 1.41 | 1.81 | |

## TABLE 5

Population Versus Concentration of Uranium Distribution for Drinking Water Sources That Serve More Than 10,000 People. The numbers in parentheses are the number of cities sampled followed by the total in the category.

| | Uranium Concentration (pCi/l)* | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| POPULATION | 0.05 -0.5 | 0.51 -1.0 | 1.1 -2 | 2.1 -3 | 3.1 -4 | 4.1 -5 | 5.1 -6 | 6.1 -7 | 7.1 -8 | 8.1 -9 | 9.1 -10 | 10./0 -11 | greater than 11 pCi/l |
| 10,001 - 50,000 (532/2407) | 292 | 75 | 46 | 14 | 17 | 4 | 75 | 4 | | 2 | | 3 | |
| 50,001 - 75000 (63/224) | 36 | 4 | 4 | 3 | 3 | | 10 | | 1 | 1 | | | (12 pCi/l) |
| 75,001 - 100,000 (31/101) | 15 | 5 | 3 | 1 | 4 | | 3 | | | | | | |
| greater than 100,000 (174/276) | 117 | 15 | 10 | 7 | 8 | 3 | 7 | 4 | 2 | | | | (30.2 pCi/l) |

+assumed secular equilibrium of U-234 and U-238.

## TABLE 6

## Standards for Radon in Air

| | |
|---|---|
| United States (Us79) | In phosphate mining regions in Florida<br>4 pCi/l take remedial action<br>2 pCi/l reduce to as low as is reasonably achievable |
| Canada (At77) | In Uranium mining regions<br>30 pCi/l take prompt remedial action<br>4 pCi/l take remedial action<br>2 pCi/l investigate |
| Sweden (Ak81) | 11 pCi/l for existing buildings<br>4 pCi/l for houses undergoing remodeling<br>2 pCi/l for newly constructed houses |

## TABLE 7

### Average Radon Values In Private And Public Water Supplies

|  | Arithmetic Mean pCi/l | Maximum pCi/l | Number of Samples |
|---|---|---|---|
| Maine (He79,83b) | 10,000 | 1,000,000 | 2,000 |
| In granite zones | 22,000 | 300,000 | 136* |
| In sillimanite grade zone | 13,600 | 100,000 | 35* |
| In chlorite | 1,100 | 2,500 | 56* |
| In public utilities | 2,000 | 11,700 | 64 |

*Rock grade determined by geologist for each private well.

## TABLE 8

## The Distribution of Radon in Municipal Water Supplies in the Central United States

| | Well Waters | | | Distribution Systems | | |
|---|---|---|---|---|---|---|
| STATE | N | G.M. (pCi/l) | G.S.D. | N | G.M. (pCi/l) | G.S.D. |
| Arkansas | 6 | 135 | 2.66 | 20 | 47 | 1.95 |
| Indiana | 10 | 151 | 2.13 | 23 | 70 | 2.20 |
| Iowa | 33 | 175 | 2.13 | 31 | 111 | 2.45 |
| Louisiana | 61 | 151 | 1.84 | 22 | 93 | 2.54 |
| Minnesota | 28 | 252 | 2.08 | 28 | 183 | 2.65 |
| Nebraska | 47 | 262 | 1.85 | 21 | 178 | 3.68 |
| New Mexico | 27 | 287 | 2.39 | 20 | 220 | 2.16 |
| Oklahoma | 7 | 117 | 1.74 | 6 | 134 | 1.17 |
| COMPOSITE | 209 | 197 | 2.10 | 174 | 115 | 2.75 |
| Texas | | | | 278 | 131 | 2.70 |

## TABLE 9

### Radon in Water Results by State and Source

Results are geometric means in units of pCi/l. Parentheses values are numbers of samples.

| STATE | PRIVATE WELL | PUBLIC WATER SUPPLY* | PUBLIC GROUNDWATER SUPPLY | PUBLIC SURFACE WATER SUPPLY ** |
|---|---|---|---|---|
| AL | 120 (22) | 8 (31) | 70 (182) | ND (8) |
| AR | 230 ( 2) | 1400 ( 1) | 12 ( 22) | ND (1) |
| AZ | -- | -- | 250 (124) | ND (6) |
| CA | 43 ( 6) | 790 ( 2) | 470 ( 15) | ND (2) |
| CO | -- | -- | 230 ( 76) | -- |
| DE | -- | -- | 30 ( 72) | -- |
| FL | 6000 (34) | 320 ( 2) | 30 (327) | -- |
| GA | 2100 ( 2) | 44 (32) | 67 (225) | 43 (2) |
| IA | -- | -- | 220 ( 85) | ND (2) |
| ID | -- | -- | 99 (155) | -- |
| IL | -- | -- | 95 (314) | -- |
| IN | -- | -- | 35 (185) | -- |
| KS | -- | -- | 120 ( 47) | 74 (2) |
| KY | 1500 (10) | ND (18) | 32 (104) | ND (5) |
| MA | 1000 ( 8) | 7 ( 2) | 500 (212) | 38 (2) |
| ME | 7000 (24) | 990 (71) | -- | -- |
| MN | 1400 ( 1) | 600 ( 1) | 130 (233) | -- |
| MO | ND ( 2) | -- | 24 (138) | ND (2) |
| MS | -- | 260 ( 2) | 23 (104) | -- |
| MT | 4300 ( 8) | -- | 230 ( 71) | ND (6) |
| NC | 15 (29) | 27 ( 2) | 79 (404) | ND (4) |
| ND | -- | 440 ( 2) | 35 (133) | -- |
| NH | 1400 (18) | 9 (12) | 940 ( 52) | ND (6) |
| NJ | -- | -- | 300 ( 38) | -- |
| NM | 59 (14) | 45 ( 8) | 55 (171) | ND (18) |
| NV | -- | -- | 190 ( 57) | -- |
| NY | 1500 ( 4) | 34 (20) | 52 (292) | ND ( 1) |
| OH | -- | -- | 79 (165) | -- |
| OK | -- | -- | 93 ( 83) | -- |
| OR | 450 (18) | -- | 120 ( 69) | ND ( 4) |
| PA | 910 (16) | -- | 380 (105) | -- |
| RI | 6500 (69) | 5200 ( 6) | 2400 (575) | ND (10) |
| SC | 1100 (28) | -- | 130 (384) | ND (14) |
| SD | 4200 ( 2) | 59 ( 2) | 210 (155) | -- |
| TN | ND ( 2) | ND ( 2) | 12 ( 98) | -- |
| UT | -- | -- | 150 (195) | -- |
| VA | 560 (42) | -- | 350 (284) | ND ( 4) |
| VT | 210 (23) | 840 ( 4) | 660 ( 71) | 13 (16) |
| WI | 730 (40) | 28 ( 4) | 150 (278) | ND (12) |
| WY | -- | -- | 330 ( 32) | ND ( 2) |
| US | 920 (434) | 68 (224) | 130 (6298) | 1 (131) |

*May include both ground water and surface

**ND - Not detected above background levels.

## TABLE 10

## Radon in Water Results by State and Population

All results are geometric means in units of pCi/l. Parentheses values are in numbers of samples.

Population Ranges

| STATE | < 100 | 100-1000 | 1000-5000 | 5000-10,000 | > 10,000 | Unknown |
|---|---|---|---|---|---|---|
| AL | -- | 83 (10)* | 59 (76) | 82 (46) | 68 (44) | 170 ( 6) |
| AR | -- | 32 ( 6) | 6 ( 3) | -- | 12 ( 2) | 8 (13) |
| AZ | -- | 240 ( 2) | 200 (68) | 350 (22) | 340 (30) | 160 ( 2) |
| CA | -- | -- | -- | -- | -- | 470 (15) |
| CO | -- | 130 ( 6) | 220 (54) | 400 ( 8) | 300 ( 8) | -- |
| DE | -- | 100 ( 4) | 39 (44) | 11 (12) | 23 (12) | -- |
| FL | -- | 320 ( 2) | 290 ( 4) | 49 (78) | 24(243) | -- |
| GA | 57 ( 4) | 42 (12) | 130 (56) | 190 (27) | 52 (45) | 39 (81) |
| IA | -- | 1120 (13) | 230 (36) | 150 (24) | 66 (10) | 75 ( 2) |
| ID | 6 ( 3) | 210 (14) | 130 (83) | 36 (25) | 100 (30) | -- |
| IL | -- | 71 ( 1) | 81 (30) | 80 (98) | 110(185) | -- |
| IN | -- | -- | 45 (71) | 17 (58) | 50 (56) | -- |
| KS | 260 ( 2) | 230 (12) | 43 (11) | 320 ( 4) | 58 ( 8) | 210(10) |
| KY | -- | 20 (10) | 42 (76) | 36 ( 8) | 5 (10) | -- |
| MA | 3300 ( 2) | -- | 380 (47) | 540 (67) | 510 (88) | 580( 8) |
| ME | 670 ( 3) | 1600 (23) | 690 (33) | 2700 ( 7) | 450 ( 4) | 400( 1) |
| MN | -- | 180 (22) | 150 (76) | 68 (43) | 140 (90) | 330( 2) |
| MO | ND ( 2) | 58 (54) | 26 (54) | 1100 ( 4) | 78 ( 6) | ND(18) |
| MS | -- | -- | 15 (45) | 51 (26) | 23 (33) | -- |
| MT | 740 ( 4) | 280 ( 6) | 270 (49) | 160 ( 8) | 18 ( 4) | -- |
| NC | 11,000 ( 6) | 250(111) | 45 (229) | 16 (32) | 21 (16) | 8100(10) |
| ND | -- | 13 (10) | 39 (112) | 97 ( 5) | 10 ( 6) | -- |
| NH | 1700 ( 2) | 1200 ( 4) | 960 (24) | 1000 (14) | 550 ( 8) | -- |
| NJ | -- | -- | -- | 360 ( 6) | 1200 (12) | 120 (20) |
| NM | 1300 ( 2) | 620 ( 7) | 48 (89) | 42 (16) | 33 (49) | 450 ( 8) |
| NV | -- | 220 ( 2) | 240 (36) | 72 ( 9) | 530 ( 2) | 180 ( 8) |
| NY | -- | -- | 56 (59) | 31 (85) | 71(113) | 56 (35) |
| OH | -- | 300 ( 2) | 160 ( 4) | 56 (76) | 100 (83) | -- |
| OK | -- | 260 ( 1) | 65 (33) | 190 (21) | 79 (26) | 96 ( 2) |
| OR | -- | 210 ( 2) | 110 (48) | 23 ( 6) | 320 ( 6) | 180 ( 7) |
| PA | 1900 ( 2) | -- | 260 (34) | 450 (32) | 440 (37) | 910 (16) |
| RI | 1700( 91) | 3900(325) | 980 (71) | 1300 (30) | 1200 (58) | -- |
| SC | 1400 (32) | 870 (30) | 92 (229) | 74 (60) | 60 (31) | 410 ( 2) |
| SD | 310 (16) | 300 (41) | 160 (85) | 210 ( 8) | 200 ( 5) | -- |
| TN | -- | 160 ( 4) | 19 (48) | 6 (25) | 5 (21) | -- |
| UT | -- | 260 ( 2) | 140 (104) | 200 (39) | 170 (48) | 150 ( 2) |
| VA | 85 ( 6) | 880 (56) | 140 (151) | 320 (12) | 720 (14) | 2300 (45) |
| VT | 120 ( 1) | -- | 540 (24) | -- | 1000 ( 2) | 750 (44) |
| WI | -- | -- | 150 (169) | 190 (61) | 130 (48) | -- |
| WY | -- | 880 ( 6) | 230 (19) | 530 ( 6) | 54 ( 1) | -- |
| US | 990(175) | 620(777) | 98(2446) | 92(1098) | 88(1464) | 140(338) |

*Number of data points used to calculate geometric mean.

**ND - not detected above background levels.

## TABLE 11

Indoor Radon Concentrations (pCi/l) in "Background" U.S. Dwellings

| Site | Number of Dwellings | Geom. Mean | Geom S.D. | Eql. Ratio | Comments | Reference |
|---|---|---|---|---|---|---|
| New York<br>New Jersey | | | | | | |
| Basements | 18 | 1.7 | 2.0 | 0.48 | Ordinary Houses | Ge80 |
| 1st Floor | 18 | 0.83 | 2.0 | 0.49 | | |
| 2nd Floor | 9 | 0.77 | 1.8 | 0.45 | | |
| Central Maine | | | | | Ordinary Houses | Pr83a |
| Basements | 77 | 2.46 | 2.4 | | | |
| 1st Floor | 82 | 1.40 | 2.4 | | | |
| Bedroom (2nd) | 81 | 1.12 | 2.4 | | | |
| Bathroom | 81 | 1.62 | 2.4 | | | |
| Outside | 67 | 0.46 | 2.4 | | | |
| Houston, Texas | | | | | Houses, Apts. | Pr83a |
| Bedroom | 103 | 0.39 | 2.5 | | | |
| Bathroom | 103 | 0.58 | 2.5 | | | |
| Outdoors | 81 | 0.22 | 2.6 | | | |
| Eastern Pennsylvania | | | | | | Sa81 |
| Living Area | 36 | | | | | |
| Summer | | 1.22 | 4.6 | | | |
| Winter | | 4.40 | 3.7 | | | |
| Basement | 36 | | | | | |
| Summer | | 3.40 | 6.4 | | | |
| Winter | | 5.90 | 4.0 | | | |

## TABLE 12

### Factor Relating Radon in Indoor Air to Radon in Water

(pCi/l In Air Per $10^5$ pCi/l in Water)

| Factor | Reference | Conditions |
|---|---|---|
| 14 | (Ka80) | Calculated value for typical Finnish single and double family structures, based on experimentally determined radon releases. |
| | (Ge80) | Calculations based on 4 occupants, experimentally determined radon releases<br>Volume Air Change Rate |
| 50 | | 15000 l 0.25 hr-1 |
| 10 | | 34000 l 0.50 hr-1 |
| 5 | | 34000 l 1.00 hr-1 |
| 4 | (Pr83a) | Observation obtained by regression from 3 months integrated data from 80 houses in Maine, causality not strictly implied. |
| < 1 | (Mc80) | Air and water grab samples in 13 structures in Halifax, Nova Scotia |

## TABLE 13

## Radon Levels Measured in Air in Houses

| | Average pCi/l | Maximum pCi/l | Number of Houses |
|---|---|---|---|
| Maine (He82b) | 3.0 | 63.0 | 85 |
| New York | | | |
| Albany Area (F180) | 3.1 | 26.0 | 21 |
| New York City Area (Br79) | 1.0 | 5.9 | 21 |
| California | | | |
| San Francisco (Ne81) | 0.4 | 1.1 | 28 |
| Pennsylvania | | | |
| Eastern (Sa81) | 10 | | 36 |
| Maryland (Mo81) | 3.7 | 27.0 | 56 |

## TABLE 14

### Radon Daughters in Swedish Dwellings

| Radon Daughters (Working Levels) | Radon (pCi/l, f=0.5) | Number of Dwellings | Percent of Dwellings |
|---|---|---|---|
| 0.000 - 0.054 | 0 - 11 | 6326 | 52 |
| 0.054 - 0.108 | 11 - 22 | 4050 | 34 |
| 0.108 - 0.270 | 11 - 54 | 1545 | 13 |
| > 0.27 | > 54 | 162 | 1 |

TABLE 15. Cases Prevented for Uranium Concentrations in Public Water

| pCi/l | % Supplies | Number Supplies | People Exposed* | Average U > | Cases |
|---|---|---|---|---|---|
| > 1 pCi | 23.6 | 10,808 | $10.8 \times 10^6$ | 7.5 | 275 |
| > 5 pCi/l | 7.88 | 3,609 | $3.6 \times 10^6$ | 17.5 | 214 |
| > 10 pCi/l | 3.46 | 1,584 | $1.5 \times 10^6$ | 30.3 | 154 |
| > 20 pCi/l | 1.33 | 609 | $0.6 \times 10^6$ | 54.8 | 112 |

*1000/supply

TABLE 16. Assessment of Water Risks for Radon in Public Groundwater

| Population | Number of Utilities Sampled | Mean Concentration Of Radon in Water (pCi/l) | | U.S. Population Using This Water (millions) | Lifetime Risk | |
|---|---|---|---|---|---|---|
| | | GEOM | ARITH | | GEOM | ARITH |
| < 100 | 88 | 990 | 6500 | 1.03 | 407 | 2678 |
| 100 - 1000 | 377 | 620 | 4100 | 7.4 | 1835 | 12136 |
| 1000 - 5000 | 1223 | 98 | 390 | 14.0 | 548 | 2184 |
| 5000 - 10,000 | 549 | 92 | 350 | 8.4 | 309 | 1176 |
| 10,000 - 100,000 | 704 | 92 | 290 | 28.3 | 1018 | 3283 |
| > 100,000 | 32 | 52 | 150 | 14.3 | 297 | 858 |
| | | | | 59.13 | 4414 | 22315 |

Population weighted Average 187. pCi/l - 944. pCi/l.

GEOM

FACTOR USED: $\frac{0.4 \times 10^{-6} \text{ deaths}}{\text{pCi/l water}}$

TABLE 17. Concentrations of Th, Pb, and Po Isotopes in Ground Water (pCi/l).

| Description | Th-232 | Th-230 | Pb-210 | Po-210 | Reference |
|---|---|---|---|---|---|
| New Mexico | | | | | |
| Grants Mineral Belt (54 wells) | | | | | |
| Paquate-Jackpile Area | <0.01 | <0.02 | | 0.39 | Ka 76 |
| Grants-Bluewater Area | <0.01 | 0-0.04 | | 0-0.66 | Ka 76 |
| United Nuclear Area | 0-0.03 | 0-0.099 | | 0.3-2.3 | Ka 76 |
| Ambrosia Lake | <0.03 | 0.0.08 | | 0-3.8 | Ka 76 |
| Gullup Area | <0.02 | 0-0.09 | | 0-0.6 | Ka 76 |
| Rapides Parish | | | | | |
| Louisiana (1 well) | | | | 290-607 | Mu 82 |
| California well | 1.3 | 1.1 | | ND | This paper |
| Arizona well | ND* | ND | | 0.9 | This paper |
| Connecticut | | | | | |
| glacial drift | | | 0.02 | <0.001 | Kris 82 |
| glacial drift | | | 0.02 | 0.004 | Kris 82 |
| crystalline rock | | | 0.03 | 0.06 | Kris 82 |
| sandstone | | | 0.12 | 0.020 | Kris 82 |
| sandstone | | | 0.07 | 0.005 | Kris 82 |
| sandstone | | | 0.03 | 0.004 | Kris 82 |
| Leesville, S.C. | | | | | |
| sand aquifer | | 0.01 | | | This paper |

ND = not detected

# Figure Captions

1. Figure 1, Map of geological provinces of the United States according to Beddinger (Be81).

2. Figure 2, Map showing the approximate locations and general areas of public water supplies which exceed 5 pCi/l of total Ra (Ra-229 was reported or combined with Ra-226 for about one-half of the sites). Large dots represent individual violations. The dot pattern represents the general area of a group of violations, with the adjacent number indicating the number of violations in that group. When the locations were unknown, just the number of violations was indicated (modified after Cothern and Lappenbusch (Co83)).

3. Figure 3, Map of population averaged uranium concentration in pCi/l for surface water in the United States.

4. Figure 4, Map of population averaged uranium concentration in pCi/l for ground water in the United States.

5. Figure 5, Map of population averaged uranium concentration in pCi/l for domestic water in the United States.

6. Figure 6, Map of geometric average radon concentration in pCi/l for public ground water supplies in the United States.

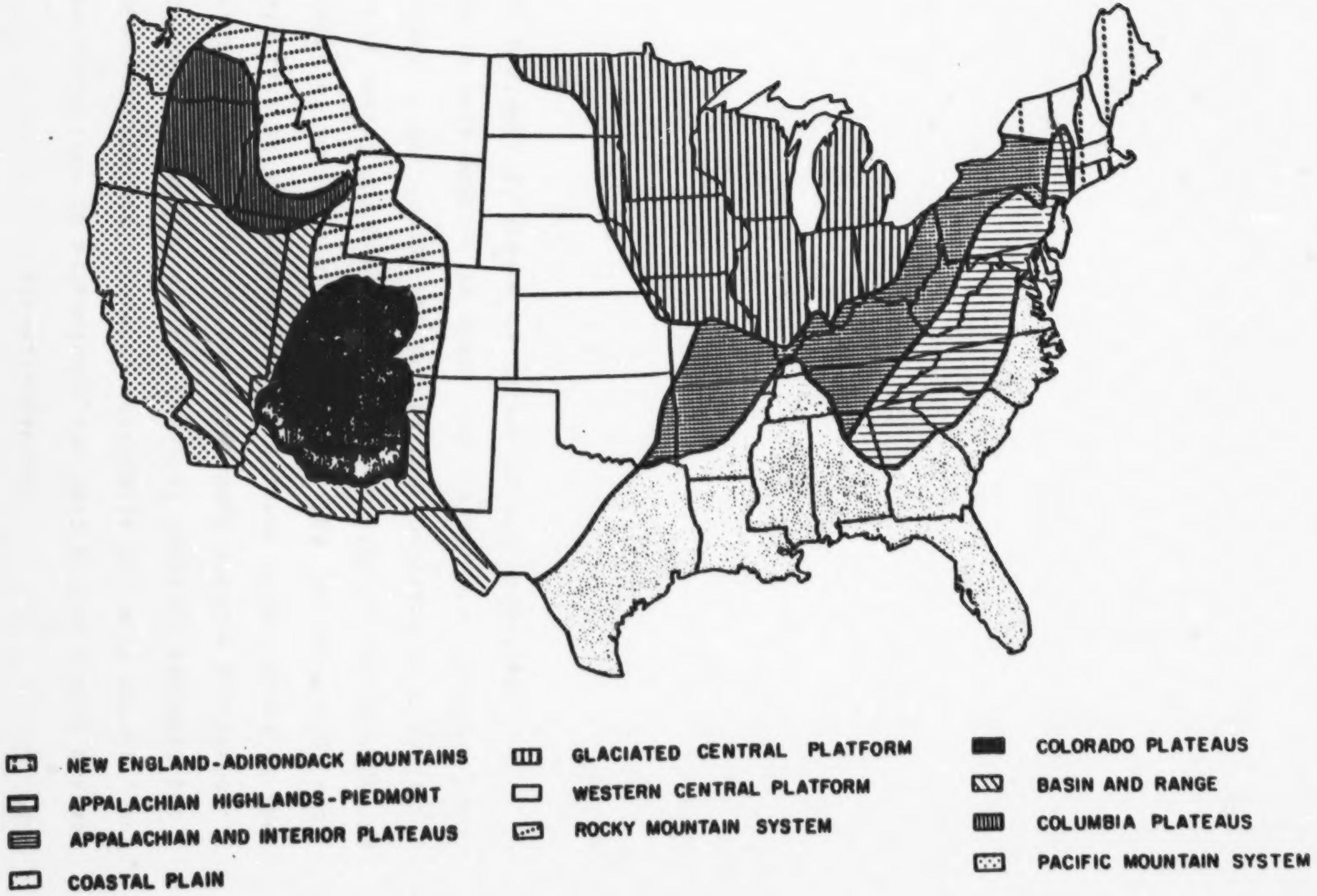
7. Figure 7, Histogram showing radium concentrations in pCi/l for public water supplies exceeding 5 pCi/l in the United States.

8. Figure 8, Histogram showing uranium concentrations in pCi/l for domestic water supplies in the United States.

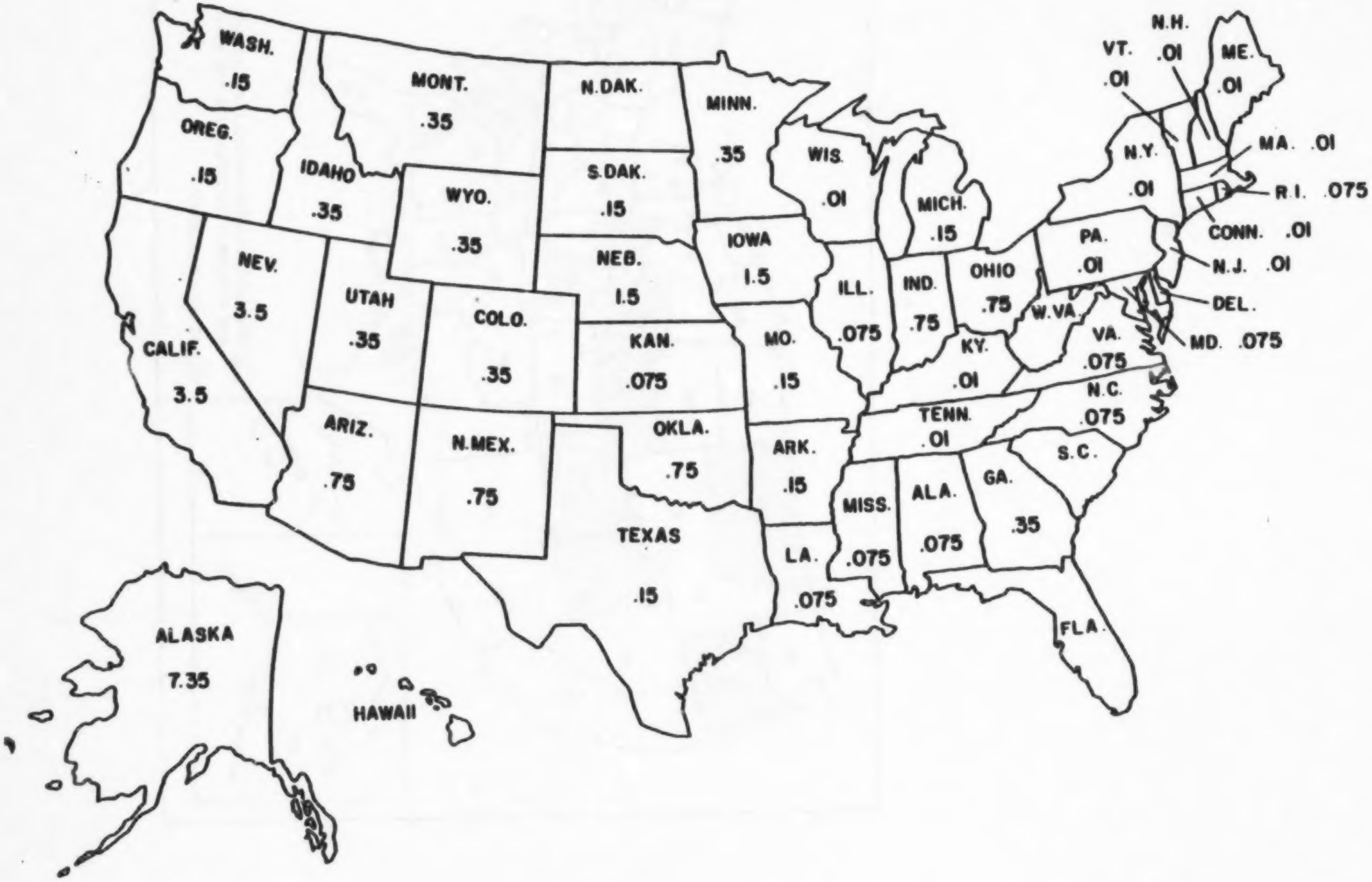
9. Figure 9, Histogram showing radon concentrations in pCi/l for public ground water supplies in the United States.

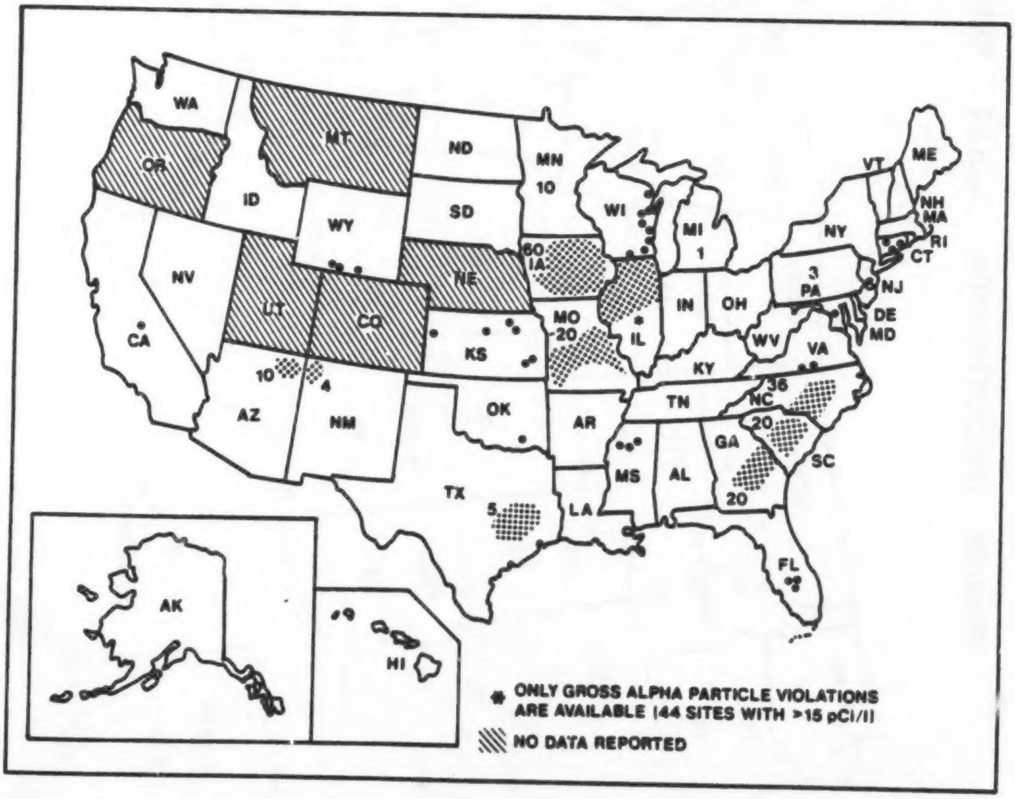
# Acknowledgements

We would like to acknowledge the help of the United States Environmental Protection Agency especially Dr. Rick Cothern, and Dr. William Lappenbusch and Mr. Drury of Oakridge National Lab for helping us to obtain information about uranium occurrence. We would like to acknowledge the assistance of Dr. Moore of the University of South Carolina for previewing the manuscript and suggestng useful changes. We would also like to acknowledge the objections of Mr. Thomas Horton, to the inclusion of the risk section in this paper. We thank the typist, Mrs. Patricia Heal for her patience in making revisions to the manuscript.

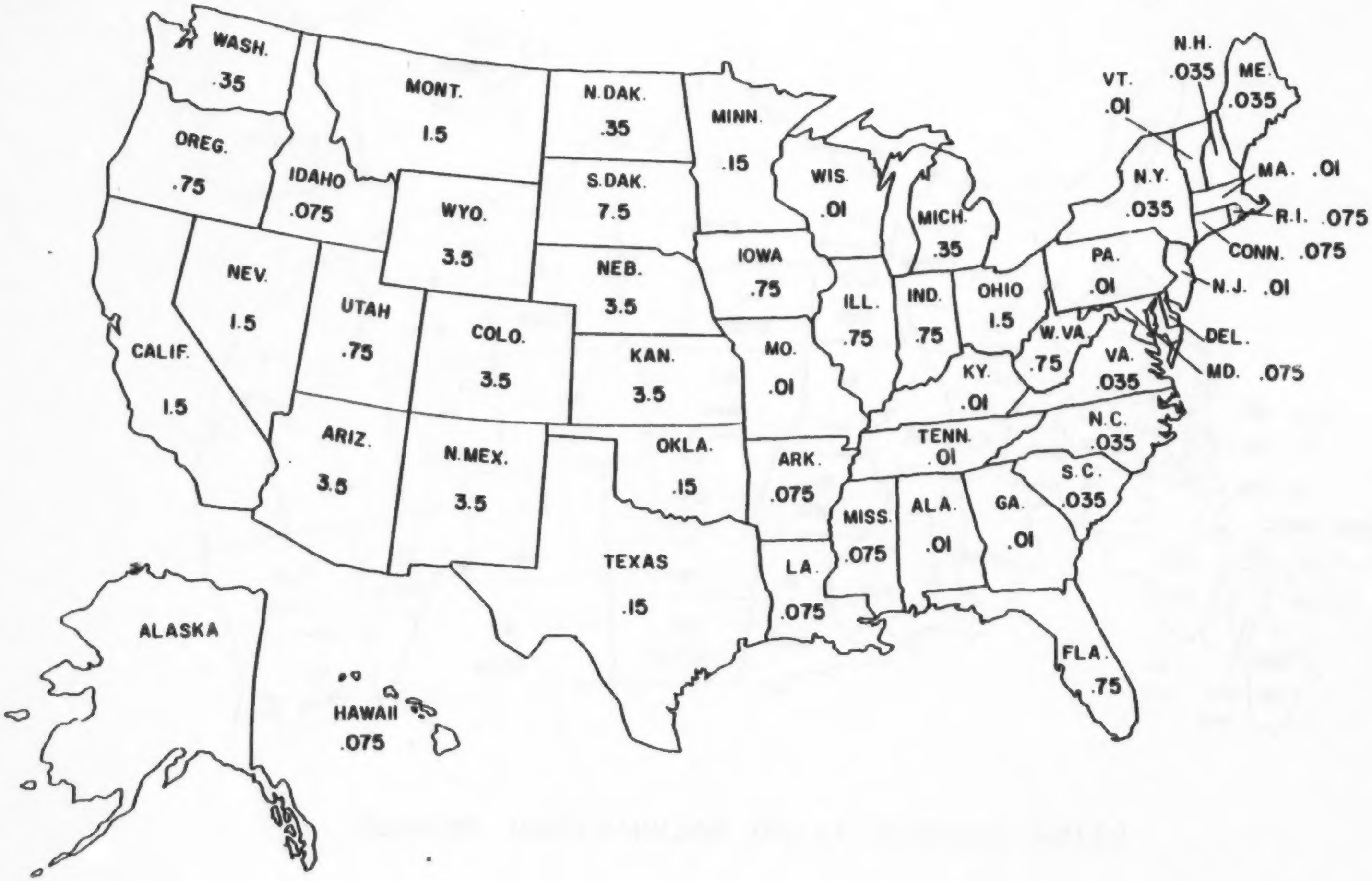

NEW ENGLAND-ADIRONDACK MOUNTAINS
APPALACHIAN HIGHLANDS-PIEDMONT
APPALACHIAN AND INTERIOR PLATEAUS
COASTAL PLAIN
GLACIATED CENTRAL PLATFORM
WESTERN CENTRAL PLATFORM
ROCKY MOUNTAIN SYSTEM
COLORADO PLATEAUS
BASIN AND RANGE
COLUMBIA PLATEAUS
PACIFIC MOUNTAIN SYSTEM

# URANIUM CONCENTRATION (pCi/l) SURFACE WATER


WASH. .15
OREG. .15
CALIF. 3.5
NEV. 3.5
IDAHO .35
MONT. .35
WYO. .35
UTAH .35
ARIZ. .75
COLO. .35
N.MEX. .75
N.DAK.
S.DAK. .15
NEB. 1.5
KAN. .075
OKLA. .75
TEXAS .15
MINN. .35
IOWA 1.5
MO. .15
ARK. .15
LA. .075
WIS. .01
ILL. .075
MICH. .15
IND. .75
OHIO .75
KY. .01
TENN. .01
MISS. .075
ALA. .075
GA. .35
FLA.
S.C.
N.C. .075
VA. .075
W.VA.
PA. .01
N.Y. .01
VT. .01
N.H. .01
ME. .01
MA. .01
R.I. .075
CONN. .01
N.J. .01
DEL.
MD. .075
ALASKA 7.35
HAWAII


WA
OR
MT
ID
ND
MN
10
SD
WY
WI
MI
1
ME
VT
NH
MA
NY
RI
CT
NV
UT
CO
NE
60
IA
IL
IN
OH
3
PA
NJ
DE
MD
CA
KS
MO
20
KY
WV
VA
10
4
AZ
NM
OK
AR
TN
NC
36
20
SC
GA
20
MS
AL
TX
5
LA
FL
AK
HI
● ONLY GROSS ALPHA PARTICLE VIOLATIONS ARE AVAILABLE (44 SITES WITH >15 pCi/l)
▨ NO DATA REPORTED

URANIUM CONCENTRATION (pCi/l) GROUND WATER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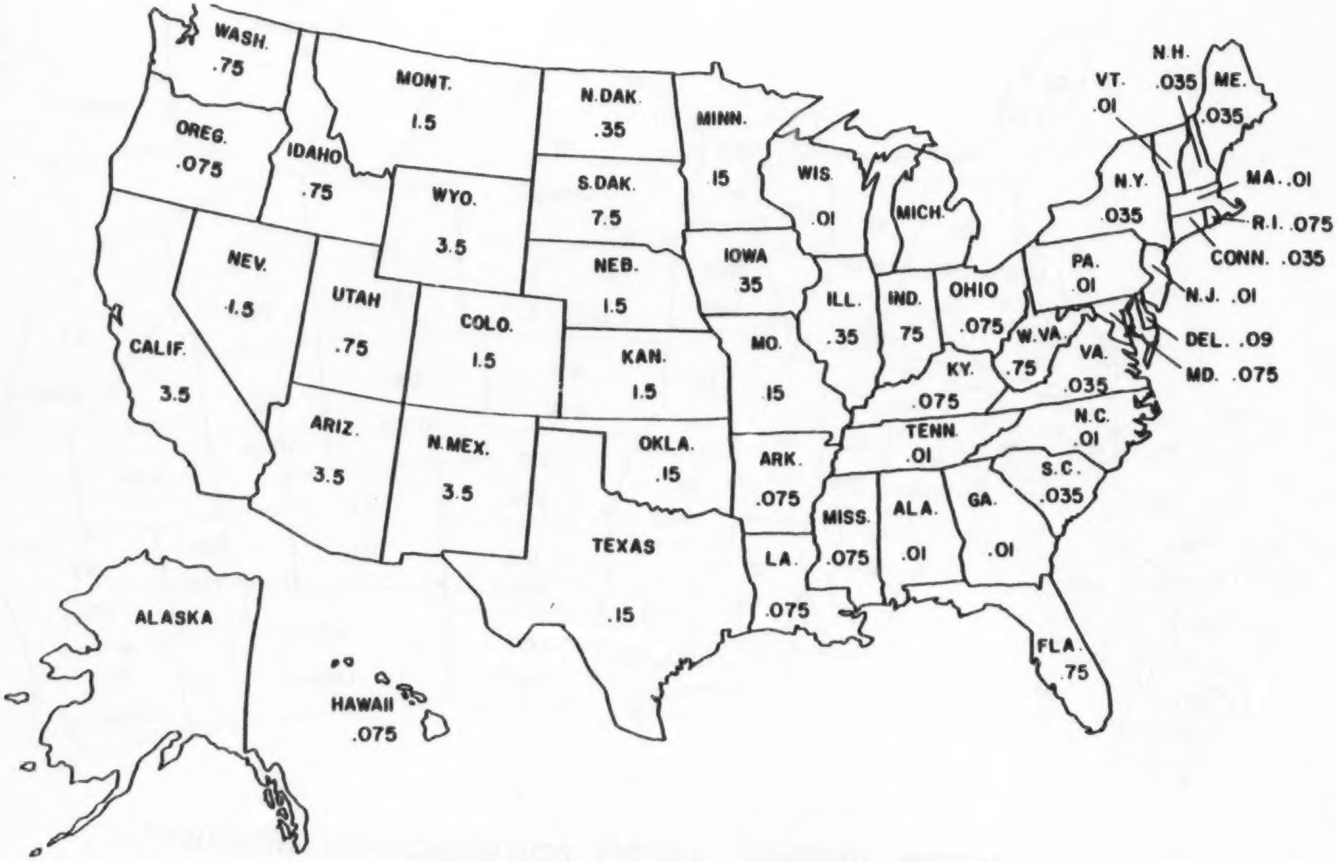
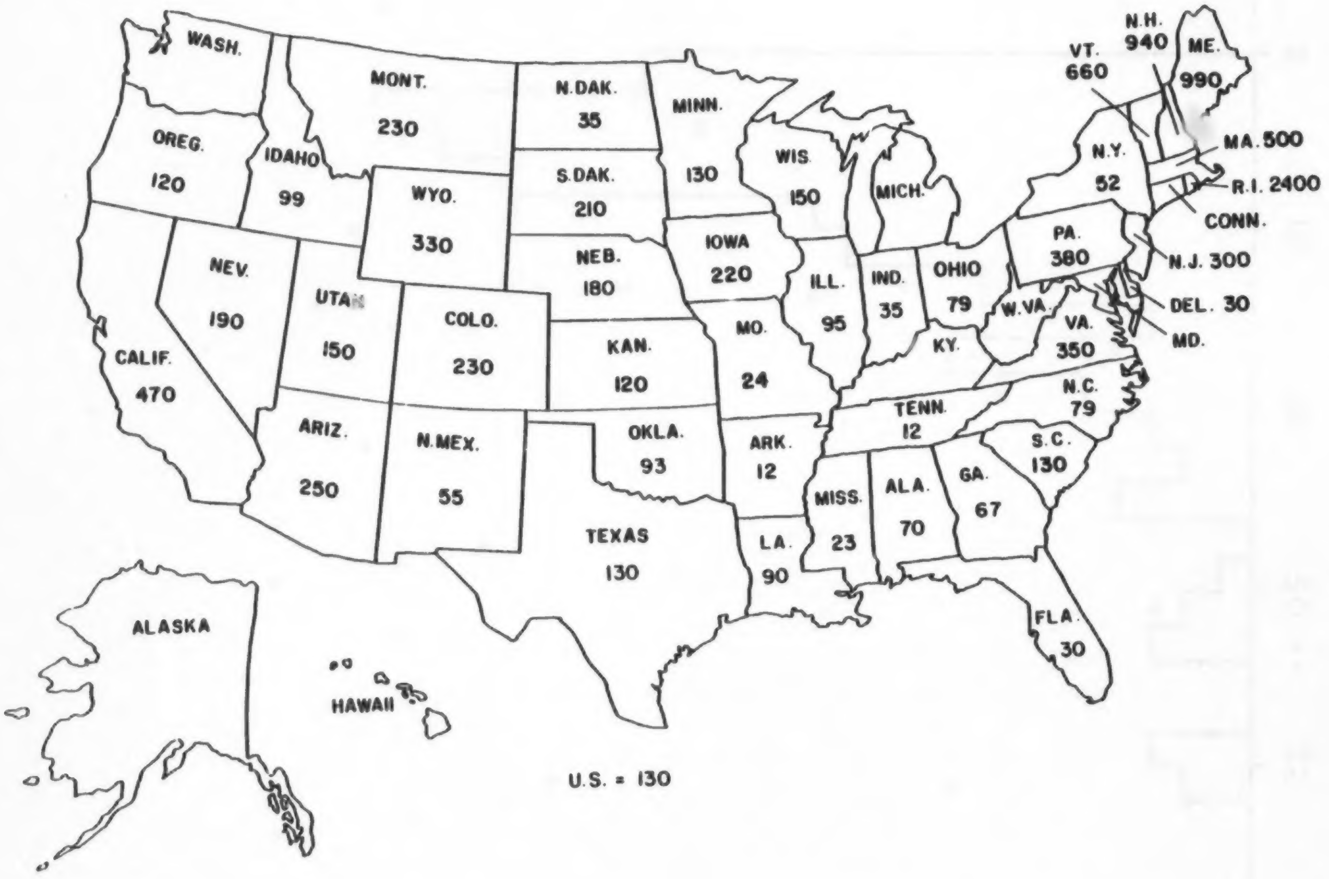
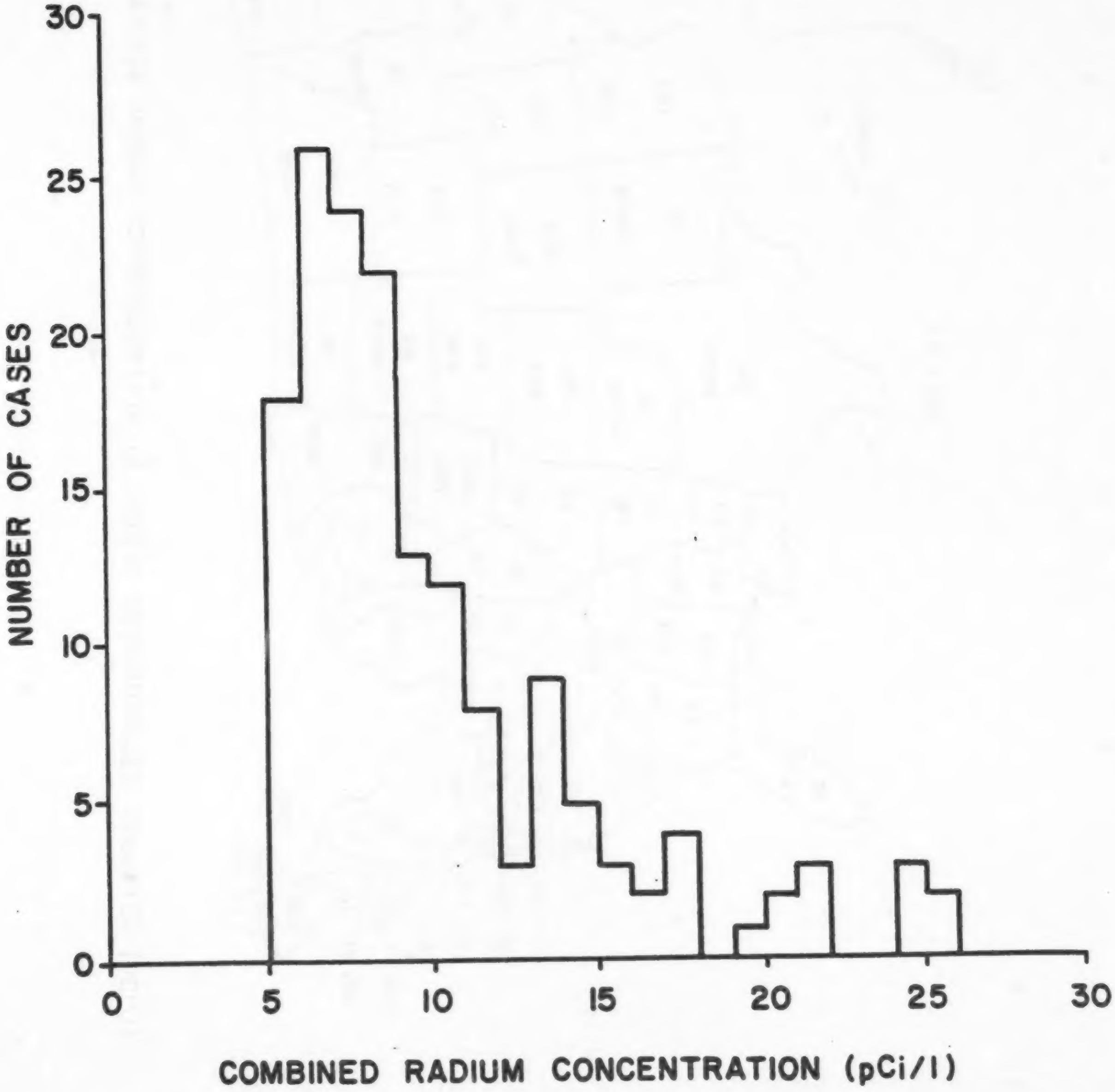
OREG. .75
CALIF. 1.5
IDAHO .075
NEV. 1.5
MONT. 1.5
WYO. 3.5
UTAH .75
ARIZ. 3.5
COLO. 3.5
N.MEX. 3.5
N.DAK. .35
S.DAK. 7.5
NEB. 3.5
KAN. 3.5
OKLA. .15
TEXAS .15
MINN. .15
IOWA .75
MO. .01
ARK. .075
LA. .075
WIS. .01
ILL. .75
MICH. .35
IND. .75
OHIO 1.5
KY. .01
TENN. .01
MISS. .075
ALA. .01
GA. .01
FLA. .75
S.C. .035
N.C. .035
VA. .035
W.VA. .75
PA. .01
N.Y. .035
VT. .01
N.H. .035
ME. .035
MA. .01
R.I. .075
CONN. .075
N.J. .01
DEL.
MD. .075
ALASKA
HAWAII .075

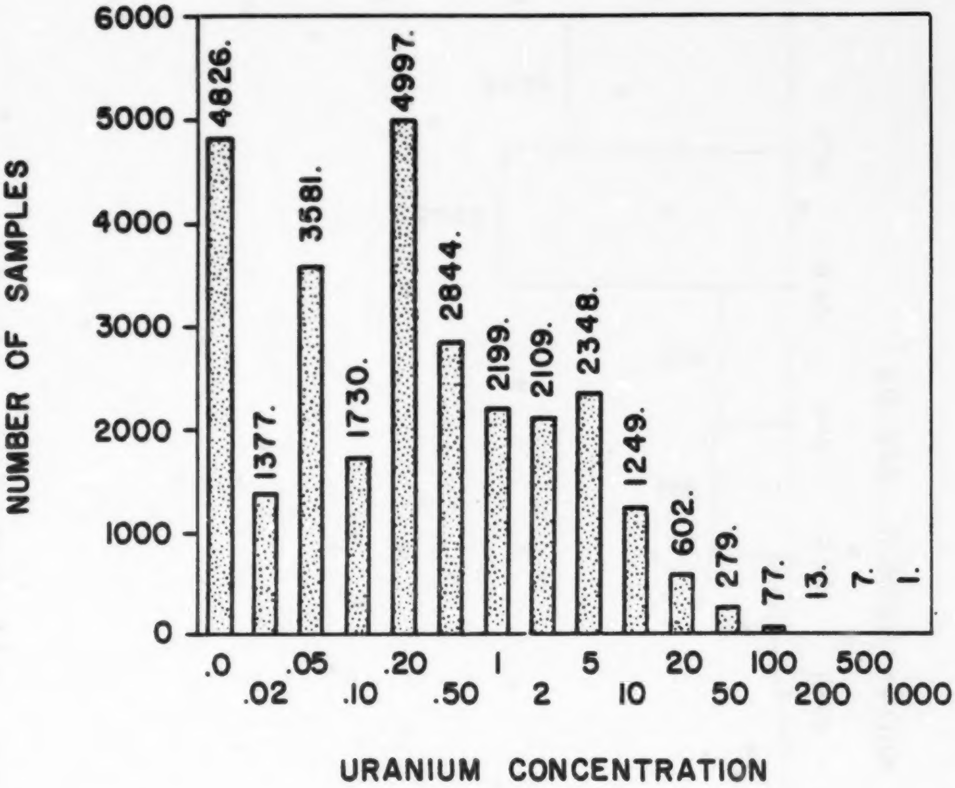
# URANIUM CONCENTRATION (pCi/l) DOMESTIC WATER


WASH. .75
OREG. .075
CALIF. 3.5
IDAHO .75
NEV. 1.5
MONT. 1.5
WYO. 3.5
UTAH .75
ARIZ. 3.5
COLO. 1.5
N.MEX. 3.5
N.DAK. .35
S.DAK. 7.5
NEB. 1.5
KAN. 1.5
OKLA. .15
TEXAS .15
MINN. .15
IOWA .35
MO. .15
ARK. .075
LA. .075
WIS. .01
ILL. .35
MICH.
IND. .75
OHIO .075
KY. .075
TENN. .01
MISS. .075
ALA. .01
GA. .01
FLA. .75
W.VA. .75
VA. .035
N.C. .01
S.C. .035
PA. .01
N.Y. .035
VT. .01
N.H. .035
ME. .035
MA. .01
R.I. .075
CONN. .035
N.J. .01
DEL. .09
MD. .075
ALASKA
HAWAII .075

GEOMETRIC AVERAGE RADON CONCENTRATION IN PUBLIC GROUNDWATER SUPPLIES (pCi/l)

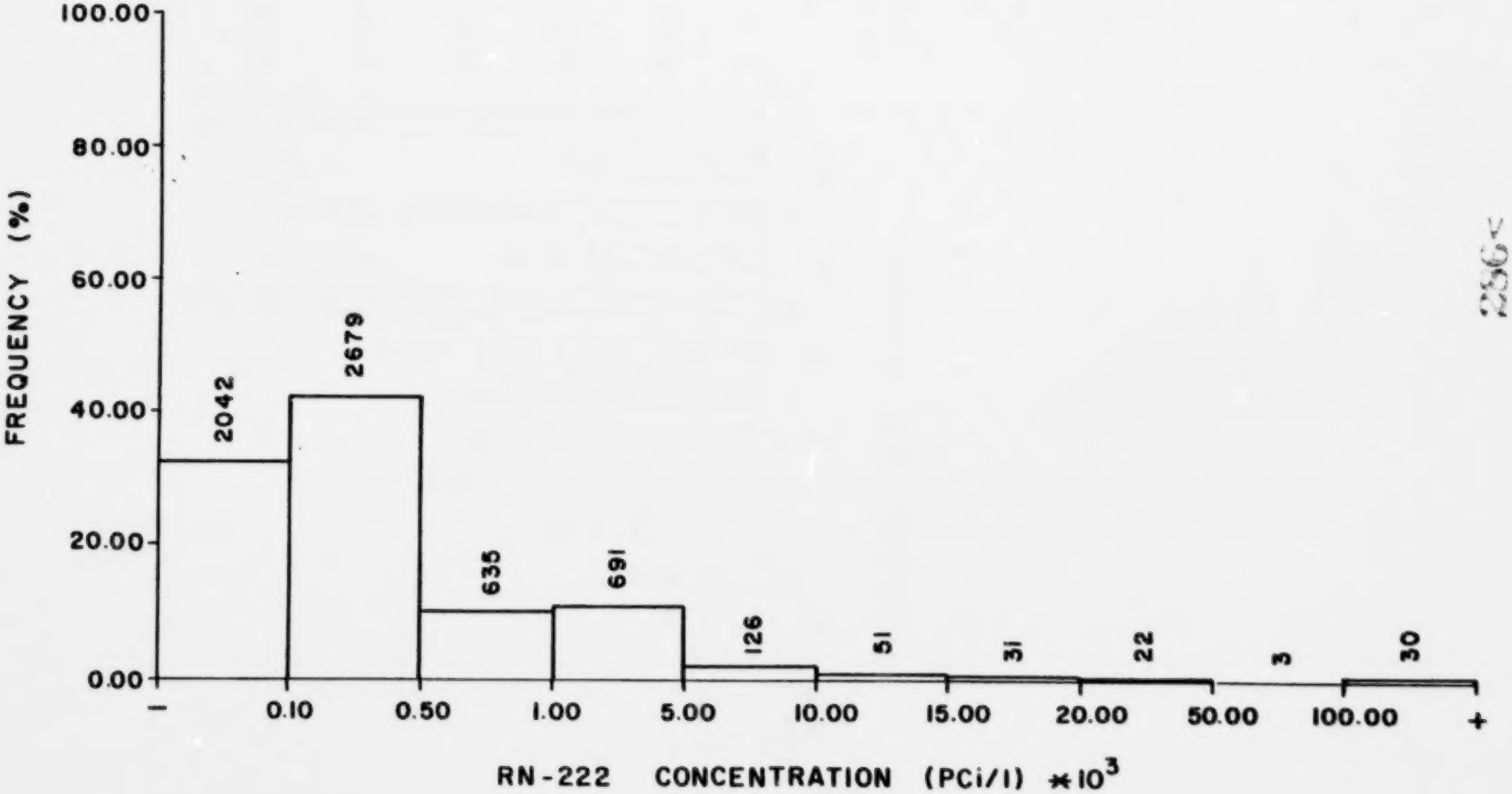

WASH.
MONT. 230
N.DAK. 35
MINN. 130
VT. 660
N.H. 940
ME. 990
OREG. 120
IDAHO 99
S.DAK. 210
WIS. 150
MICH.
N.Y. 52
MA. 500
R.I. 2400
CONN.
WYO. 330
IOWA 220
PA. 380
N.J. 300
NEV. 190
UTAH 150
NEB. 180
ILL. 95
IND. 35
OHIO 79
W.VA.
DEL. 30
MD.
CALIF. 470
COLO. 230
KAN. 120
MO. 24
KY.
VA. 350
TENN. 12
N.C. 79
ARIZ. 250
N.MEX. 55
OKLA. 93
ARK. 12
S.C. 130
MISS. 23
ALA. 70
GA. 67
TEXAS 130
LA. 90
FLA. 30
ALASKA
HAWAII
U.S. = 130


30
25
20
15
10
5
0
NUMBER OF CASES
0
5
10
15
20
25
30
COMBINED RADIUM CONCENTRATION (pCi/l)



NUMBER OF SAMPLES
6000
5000
4000
3000
2000
1000
0
4826.
1377.
3581.
1730.
4997.
2844.
2199.
2109.
2348.
1249.
602.
279.
77.
13.
7.
1.
.0
.02
.05
.10
.20
.50
1
2
5
10
20
50
100
200
500
1000
URANIUM CONCENTRATION

## PUBLIC GROUNDWATER SUPPLIES (ALL)



# COMMITTEE ON METABOLISM AND DOSIMETRY OF HIGH LET RADIONUCLIDES

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David L. Willis

# METABOLISM OF INGESTED URANIUM AND RADIUM

McDonald E. Wrenn
Radiobiology Division
University of Utah
Salt Lake City, Utah 84112

Patricia W. Durbin
Lawrence Berkeley Laboratory
Berkeley, Ca. 94720

Blaine Howard
Division of Environmental Health Services
Utah State Health Department
Salt Lake City, Utah 84110

Joyce Lipsztein
Comissao Nacional de Energia Nuclear
Instituto de Radio Protecas E Dosimetria
Avenidadas Americas, KM11,5
Rio de Janeiro, Brazil 22600

John Rundo
Argonne National Laboratory
Argonne, IL 60439

Edwin T. Still
Environment and Health Management Division
Kerr-McGee Corporation
Oklahoma City, OK 73125

David L. Willis
Department of General Science
Oregon State University
Corvallis, OR 97331

## Abstract

The literature on metabolism of U and Ra for man relevant to deriving drinking water standards has been reviewed and summarized. Ra is well understood, but significant gaps remain in our knowledge about U metabolism. Limits should be based on an equilibrium model where a constant relationship between intake and organ burden is established, using the best and most likely metabolic parameters. For the skeleton we conclude that the best estimate of skeletal burden expressed in days equivalent intake are 25 days for $^{226}$Ra, 10 days for $^{228}$Ra, and 0.3 days for $^{224}$Ra. For longlived isotopes of U, we chose 11 days, with a range between 1 and 35 days. The committee believes that intake of natural U in water should be limited by considerations of toxicity to the kidney, and we believe that the metabolic* model of Spoor and Hursh with a modified GI absorption (1.[illegible]%) should be used to infer kidney content. Our review and analysis of the world literature lead us to believe the average human GI absorption of U is most likely 1 to 2% and is probably reasonably independent of age or the mass of U ingested. Using a safety factor of 50 to 150, the committee recommends a limit of U in water of 100 micrograms/liter in order to limit toxic effects in the kidney. 100 micrograms/liter is equivalent to 67 pCi/liter of longlived alpha-emitting natural U isotopes. Further research into the distribution of U in the human body is desirable, especially at natural levels in kidney and skeleton, the time dependent pharmacokinetics of U in animals, the GI absorption of U in man from water and food, toxicological and U distribution studies in animals under conditions of chronic oral U intake, and metabolic model error propagation.

---

* In most cases throughout the text and appendices, "metabolism" can be used interchangeably with "biokinetics".

## Introduction

The metabolic models for U and Ra which are described in this section are required to estimate the risks to human health from ingesting these elements in drinking water. Chemical toxicity, which is relevant to U in its natural, depleted or slightly enriched state, is addressed, as are the radiotoxicity and the radiobiological effects of the important alpha-emitting isotopes of Ra, including $^{224}$Ra, $^{226}$Ra, and $^{228}$Ra.

Although no radiobiological effects from injected or ingested natural U have been, or probably can be, observed in occupationally exposed populations or in experimental animals, this paper estimates the kinetics of skeletal U deposition, so that risk coefficients for bone cancer induction developed by the subcommittee on risk can be applied (Mays84). This procedure utilizes the average dose to bone, rather than endosteal dose or dose to bone marrow. Dose to bone marrow is not used because populations exposed to $^{226}$Ra + $^{228}$Ra and $^{224}$Ra developed no additional leukemias above expectation (Spi83). However, for these radionuclides, many bone sarcomas were observed, indicating that dose to red marrow is less effective in inducing leukemia than the dose to bone surfaces is in inducing bone sarcomas. Skeletal cancer will be regarded as the major potential radiobiological effect of ingested alpha-emitting radioisotopes of Ra and the presumed radiobiological effect of U, if any.

Finally, best estimates of normal U metabolism will be used, because even in extreme cases the amounts of U or Ra ingested in potable water are not great enough to chemically or radiobiologically modify their metabolic behavior (Co83a). With best estimates, known factors of safety can be introduced at the ends of the assessments.

## Scope of Review

This paper cites more than 120 references, many of them review articles which, in turn, summarize large numbers of scientific papers. Periodically since 1958, and most recently in 1982, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has summarized what is known about the levels of Ra, U, other naturally-occurring radionuclides and manmade alpha-emitters in air, food, water, soils and rocks, as well as inhalation and ingestion of these materials in food and water and the relationship of that intake to their accumulation in the body (UN58, 62, 66, 69, 72, 77). Earlier, the Manhattan Project investigated and reported on the potential chemical toxicity of U in the National Nuclear Energy Series published in 1949 and 1953 (Ta51, Vo49, Vo53). Health and safety data for U in the workplace were published in 1958 (HA58) and in 1975 (Wr75). The toxicology and metabolism of U in man and animals, as well as the relevance of that data to established protection limits, were thoroughly reviewed in 1973 (Hod73a,b; Hu73, Sp73, Yu73). The metabolism and biological effects of Ra have been examined in a number of important articles (Ev66, ICRP73, Ro78, Ro83), and constitute a large part of the first supplement to Health Physics (Vol 44, Suppl. 1, 1983). The metabolism of environmental levels of Ra and U was also reviewed in a symposium on high natural background areas (Wr77b). The comparative distribution of U among lung, liver, bone and kidney, as well as local distribution in bone, was described in a followup symposium (Wr82).

In view of the availability of so much data, (especially for $^{226}Ra$), the scope of the discussions of Ra and U metabolism in this paper will be limited. Interim drinking water standards are presented in terms of concentration (amount per unit volume of water). In that the issue under consideration is ingestion, such a restricted form of expression of limits is believed to be valid, even though the underlying assumptions about the amount of daily water intake must be consistent with the objectives of the risk limitation procedure. Recommendations will be made in a form relevant to isotopes of U and Ra likely to be present in soluble form in drinking water, and it follows that water samples should be filtered prior to analysis for compliance.

The criterion we adopt to control radiological risk is to limit the likelihood of bone cancer induction in populations, not in individuals. It follows from the adoption of this criterion that the number of interest is the average intake in the population on a per capita basis, rather than a maximum intake for a maximally exposed individual in the population. Therefore, this paper uses best estimates of average intake rates of water for input to the metabolic model. The procedure developed should be sufficient to limit per capita lifetime risk to $10^{-4}$ for bone sarcomas and carcinomas in soft tissues adjacent to bone (head sinus carcinomas).

There are no data on experimental induction of bone cancer by ingested, injected or inhaled natural U in soluble form. Bone cancer has been experimentally induced in animals by U, but only higher specific activity U isotopes or mixtures of U isotopes: $^{232}U$ or $^{233}U$ injected as $UO_2(NO_3)_2$ into mice (Fin53); enriched $U^{+6}$ (90% $^{234}U$ and $^{235}U$ by weight) intratracheally instilled in rats (Fil78); $^{232}U$ or $^{233}U$ inhaled as $UO_2(NO_3)_2$ by rats (Bal83). No bone cancers or leukemias have been reported among the large numbers of rats, rabbits or dogs that were injected with, fed, or inhaled soluble or insoluble U compounds (Vo49,53; Fin53; Yu73).

For collective dose and risk, we will consider exposures leading to cumulative bone tumor risk of 10% of the natural incidence of bone cancer, or an approximate per capita lifetime risk of $10^{-4}$ in man, while noting that total "natural" cancer mortality lifetime risk in the U.S. is about $1.7 \times 10^{-1}$.

The International Commission on Radiological Protection (ICRP77a,b) has suggested that "a risk of cancer in the range of $10^{-6}$ to $10^{-5}$ per year would be likely to be acceptable to any individual member of the public." Over a 70-year lifetime, it corresponds to a lifetime risk in the approximate (because of uncertainties in latency periods and "plateau" effects) range of $7 \times 10^{-5}$ to $7 \times 10^{-4}$.

## Choice of a Metabolic Model

Radiation dose from internally deposited radionuclides is rarely obtained directly, particularly at environmental intake levels, because of the near impossibility of in situ measurements, particularly of alpha particles. Instead, other quantities are measured from which doses can be inferred. The closer the relation between the measured quantity and the dose, the fewer are the assumptions required, and the more realistic the estimate of dose is likely to be. Thus, dose is best calculated from knowledge of the amount and distribution of radionulcides in organs, which in turn may be inferred from either direct in vivo measurements or metabolic models relating intake to accumulation, distribution and excretion.

A number of mathematical models have been developed which relate body U or Ra content to intake (ICRP59, 67, 72, 79). Most of these are not easily adaptable to conditions in which both the intake and the dose rate are chronic, as in a natural environment. An exception is the model developed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UN62, 66, 69, 72) which was used to estimate radiation doses from chronic intakes of naturally occurring radionuclides.

Important considerations for determining body content of a radionuclide after it is ingested ingestion in water are time and age dependence of the intake. For purposes of the present analysis, it is assumed that organ concentrations remain constant at the equilibrium value of an adult with fixed daily intake. Of course, this is an oversimplification, since dietary composition and source of water supply can change with age and residence, as can some of the relevant metabolic parameters. However, there is evidence that such a simplification is appropriate for $^{226}$Ra, $^{228}$Ra, $^{210}$Pb, and U (Wr77b). Finally, one needs to consider the interval between conception and birth, when the accumulation of minerals depends on (a) the mother's intake, (b) the reservoir of elements in the mother's body, and (c) the ability of the placenta to discriminate between essential and non-essential (but chemically similar) elements.

In the equilibrium model, skeletal concentration is a constant and there is no dependence on age. This is relevant to a substance which may experience discrimination by the GI tract, but not by the placenta (Wr77b). In this case, the concentration of an element in the fetal skeleton should reflect that in the mother, and the ratios of the element to calcium in the newborn and in the maternal blood should be the same. Elements which satisfy this condition are the alkaline earths, which include the longlived Ra isotopes and U, which at physiological pH is in stable 6+ state as the divalent cation $UO_2^{+2}$.

The approaches we will take are to identify empirical relationships between equilibrium concentrations in the environment and the human body, or to adduce sufficient information about metabolism to predict the uptake and concentration in the body as a function of time. The latter approach will be restricted to the case of U in the kidney. There may be circumstances which alter the proportionality constant between intake and equilibrium amount in the body; for example, chemical congeners in food may influence (usually reduce) transport across the GI tract.

## Metabolic Model for Ra

This section examines placental discrimination and variation of Ra concentration in bone with age. The normal intake of Ra has been the subject of several reviews: food is normally the major source of intake (UN62, 66, 69, 72, 77). Whenever the concentration of either $^{226}Ra$ or $^{228}Ra$ exceeds several pCi/l in water, water may be the dominant source of intake. Generally, air is not an important contributor. The UNSCEAR examined normal and elevated intake of Ra in air, water, and food, and its content in the human body in the reports issued in 1958, 1962, 1966, 1972 and 1977. The following discussion draws heavily on the UNSCEAR summaries. The ICRP report entitled "Alkaline Earth Metabolism in Adult Man" (ICRP73) focused on the metabolism of transient intakes in both the occupational and medical Ra cases. The dosimetry of Ra and other alpha-emitters in bone was summarized by Spiers (Spi68) in his monograph on internal emitter dosimetry, and by the National Council on Radiation

Protection monograph on "Natural Background Radiation in the United States" (NCRP75).

The limited evidence shows that under conditions of chronic intake the concentration of longlived naturally occurring alpha emitters per gram of body ash is nearly invariant throughout life (Mayn61, St64, Ra65, Bo63). Mayneord showed this is specifically correct for $^{226}$Ra. Thus, the natural $^{226}$Ra/Ca ratio in the human skeleton does not change (St64) from 4 months of fetal life (organogenesis is not complete until 3 months) through old age. Pisenne reported measurements of Ra in human bone which are consistent with the hypothesis that Ra in the skeleton is reasonably age-independent (Pi79). Accordingly, the model of an age-independent Ra/Ca ratio in the skeleton appears to be appropriate for continuous Ra intake (UN66).

The observed ratio in bone and diet is defined as:

$$\text{OR (bone/diet)} = \frac{\text{g Ra/g Ca in bone}}{\text{g Ra/g Ca in diet}} .$$

The OR is considerably less than unity for Ra.

A global mean OR of 0.024 or median OR of 0.020 for $^{226}$Ra is suggested by UNSCEAR in its 1977 survey of the worldwide measurements (UN77). This survey presents the results of eight studies in six countries in which the mean OR ranged from 0.013 to 0.039. The highest single value (0.039) was for San Francisco; the second highest U.S. value (0.024), for New York City. In any given study, sampling or other errors which result in a high estimate in bone or a low estimate in diet, or the failure to include significant non-dietary sources of Ra, would lead to an overestimate of OR. Using the global average intake of 0.92 pCi/day, which lies between the two U.S. values, and a mean concentration of 5.1 pCi/kg in bone (mass of mineralized tissue is 5 kg for Reference Man*), leads to a net burden in the skeleton that is equivalent to 28 days intake with a range from 20 to 37 days. Considering the potential variability of the data from which these numbers are derived, this range seems small indeed.

However, in order to make the best estimates of the Ra content of the human body from dietary information, the Ca intake should be known as well, as pointed out by Penna-Franca et al. (Pe65).

For $^{226}Ra$, it is possible to measure the body burden in vivo at low natural levels (i.e., about 20 pCi) by use of the breath radon technique, provided that care is taken to have the subject breathe radon-free air for a sufficiently long period. Stehney and Lucas (St56) measured body burdens of $^{226}Ra$ using the breath radon technique; intake rates of $^{226}Ra$ were determined for the same population by measuring $^{226}Ra$ in feces, which should give a good indication of total intake. Measurement of Ra excreted in feces avoids the need to reconstruct representative diets; such reconstruction introduces errors if foods and water are not included in their proper representative proportions. Food preparation may also alter the Ra content of the water used (e.g., in brewing coffee). Four groups were measured as shown in Table 1. The Stateville samples were from a prison having high $^{226}Ra$ in water, and the subjects were presumed to have had a constant intake during their incarceration. Since incarceration represented a change, most likely to higher $^{226}Ra$ intake, those subjects were not in equilibrium. The elevated body burdens of

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* The 5000 g of mineralized bone is 12% water, 23% organic, and 65% ash by weight; the dry mass is 4400 g (ICRP:4).

$^{226}$Ra were not established rapidly, and between $10^3$ and $10^4$ days was required to approach equilibrium.

Stehney and Lucas' evaluations of "days of intake equivalent body burden" range from 17 for the Stateville group (11 subjects with a mean time at Stateville of 20 years, probably not in complete equilibrium) to 45 days for eight boys from Lockport, Illinois (ages 15 to 18). With the exception of the latter group, the results are consistent with a value of 23 days derived from the alkaline earth model adopted by the ICRP (ICRP73). Further, assuming dietary intakes of 1 g Ca/day, the results are consistent with an OR(bone/diet) of 0.02.

As is well known from animal studies, although Ra initially deposits on bone surfaces, especially in areas of rapid bone formation, continual intake and bone remodeling cause the Ra to become uniformly distributed throughout bone.

In the past, between 10% and 30% of the $^{226}$Ra in the adult human body was believed to be in soft tissues. The ICRP model of alkaline earth metabolism suggested that 15% is an appropriate estimate under chronic exposure conditions (ICRP73). Schlenker et al. recently adjusted the ICRP model on the basis of data on the $^{226}$Ra content of soft tissues from 17 subjects who received Ra by injection or ingestion 5 days to 53 years before measurement (Sc82). They concluded that soft tissue retention peaks at 58% of the whole body retention 18 days after a single intake and then falls steadily to 33% at 100 days and 6% at 1000 days. Under conditions of chronic intake, 5.5% of the body's Ra content would be in soft tissue.

In summary, the global average approaches 28 days equivalent intake of $^{226}$Ra in the body (UN77). The ICRP alkaline earth model predicts 24 days, and the data on measured human intake rate and accumulation range from 17 to 45 days (St56). Since the alkaline earth model includes biological knowledge not inferrable from empirical measurements of $^{226}$Ra in ingesta and bone, it is reasonable to select 25 days intake equivalent as a best estimate of the equilibrium content of Ra in the skeleton.

The days equivalent intake for $^{228}$Ra and $^{224}$Ra will be inferred using parameters adopted by ICRP for the alkaline earth model. $^{228}$Ra ($T_{\frac{1}{2}}$ = 5.77 years) is ubiquitous in soils, rocks, foods, and water, and it is metabolically the same as $^{226}$Ra. $^{224}$Ra accumulation is limited by its short physical half-life of 3.62 days; it is commonly associated with $^{228}$Ra via $^{228}$Th.

We can deduce values for the body contents of $^{228}$Ra and $^{224}$Ra in units of their daily intakes, from their effective retention integrals given in Table 36 of ICRP Publication 20 (ICRP73) or in Table 8 of reference Sc82, leading to equilibrium values of body contents of 10 and 0.3 times their daily intakes, respectively (GI absorption taken as 0.2). The $^{224}$Ra will be distributed on bone surfaces, but will have insufficient time before decay to become well distributed throughout bone volume. For these parameters, the best estimate of the equilibrium Ra content in human skeleton is given in Table 2.

## Metabolic Model for U Isotopes

The metabolism of U has not been as well studied as that of Ra. Details about the gastrointestinal absorption of U and its distribution in the body are reviewed thoroughly in Appendices A and B, the results of which are summarized below.

The first topics considered are placental discrimination against U and the age-dependence of U concentration in bone. There is evidence that some U is accumulated in bone before birth. Sikov and Mahlum (Si68) studied placental transfer of U ($^{233}$U injected as citrate) in rats. The concentration of $^{233}$U in placenta relative to that in the fetus was 1.7 at 15 to 20 days of gestation. No measurements for calcium were reported. When Sikov (personal communication) compared placental transport of a number of nuclides at 19 to 20 days gestation, U showed the lowest placenta-to-fetus ratio (1.7), while the $^{137}$Cs ratio was 2.0. This animal work suggests that U/Ca ratio in fetal bone relative to maternal bone may be close to unity.

Masuda _et al._ (Mas71a,b,c,d) investigated four villages in Japan where the intake of U varied between 1 and 9 μg/day. Their work showed that urinary excretion of U appeared to be nearly independent of age after puberty. This provides presumptive evidence that U in the body is in equilibrium with intake, and the absence of placental discrimination against U relative to calcium would imply that this equilibrium is established early in life, as is the case for $^{226}$Ra.

The distribution of U in the body of Reference Man is summarized here and given in detail in Appendix B. Animal studies indicate that the amount of soluble U accumulated internally is proportional to intake from inhalation and/or ingestion.

Natural levels of U in human bones collected in the U.S. were measured in 9 studies without regard to geographical location sampled, sample treatment, or part or number of skeletons sampled; the range of skeletal U content calculated for Reference Man is 2.3 to 61.6 μg, with median and mean values of 12.9 and 24.9 μg, respectively.

Measurements of U in soft tissues are sparse. The natural U concentration in kidney appears to be only about twice that in liver, and less than that reported for some other soft tissues (i.e., fat, lung, gonads--see Table B-2). Kidney contains about 0.3% of the U estimated to be in the body of Reference Man.

The best available value for the normal U content of Reference Man determined from measurements is about 38 µg, of which 66% or 24.9 µg is in the skeleton. The proportions in bone and soft tissue are in reasonably good agreement with the results of experimental U administrations to large animals (Du75, Ste80), and human subjects (Ber57). If one estimated dietary U intake at 1.75 µg U/day, then there is 14 days equivalent U intake in skeleton. In Appendix C, kinetic modeling of U uptake in bone, using the ICRP model with modified GI uptake and modified parameters for U distribution in bone compartments, gives 11 days equivalent accumulation of U in skeleton.

Neutron-induced autoradiographs of natural U in bone show that under equilibrium conditions it is diffusely distributed throughout the bone volume (Sc73, Wr82). Higher than average U concentrations are found on bone surfaces shortly after a single intake (Ro68; Ste80), but the U gradually assumes a pattern which is more diffuse. Neutron track autoradiographs of bones from dogs exposed only to environmental U in the diet show uniform distribution. Under chronic exposure conditions, U in human bone is likewise expected to be reasonably uniformly distributed throughout the volume.

GI absorption studies of U include single oral administration experiments of soluble uranyl compounds to rats, dogs, hamsters, and a baboon, continuous feeding of dry salts to adult rats, and single administrations of soluble salts to neonate rats and swine. GI absorption is consistently lower in the rat than in the other animals studied. For this reason, the data on GI absorption in the rat should not be used uncritically to infer human absorption. There is some evidence that GI absorption may decrease very slowly with increasing mass of U fed (see Figure A-1).

There are four sets of data dealing with measurements of U in diet and excreta of man which can be used to infer average GI absorption from food and water. There are six sets of animal experiments which are suitable for the same purpose, for a total of 10 suitable experimental estimates of GI absorption of soluble U over a range of U intakes from $2.5 \times 10^{-2}$ to $6.7 \times 10^{3}$ μg/kg/day. The mean values range from 0.3 to 7.8% of ingested U absorbed, with a mean of 1.6%. If the single highest value, 7.7%, is excluded, the mean absorption is 1.1%. Least squares fits of linear, semi-log and log-log functions to GI absorption vs. dosage (μg/kg) yielded U absorptions at natural intake levels in the range of 1.3 to 1.9%. The highest single value of U absorption, 7.7%, comes from coupling an older study of urinary U excretion in man and a U.S. dietary survey and measurement program (fluorescence analysis of U was used for both studies). Because of limitations on sensitivity of the fluorometric technique for the urinary measurements and the tenuous relationship between the urine U levels in a few persons and the U content of their diet (inferred from a small-sample diet survey), this value is the most doubtful of those available. For this evaluation, we believe that the value for U absorption obtained in that way may properly be excluded in inferring GI absorption in man. The power function fit to the nine other studies gives a GI absorption of 1.4% at environmental levels of U intake, and may decrease slightly with increasing U intake. A value of 1.4% is adopted as our best estimate for subsequent analysis.

The multiple compartment U metabolic model of Struxness (Str55) was improved to include a two-compartment skeleton (Wr77a, Wr78, Ros80). In addition, an elegant direct solution of a five-compartment model by Lipsztein et al. (Li81), a blood-organ transfer kinetic model by Skrable et al. (Sk80), and the non-connecting models of Spoor and Hursh (Sp73) and ICRP (ICRP79), which have no feedback through blood, have also been developed. Not only the models, but also the best values of the parameters to insert in the models, vary with author preference and the kind and quality of the biological data available to them. All of the models cited, using the biological parameters preferred by their authors, give results for equilibrium skeletal U accumulation in the range of 1 to 40 days of intake. Calculations of the predicted equilibrium concentrations in human kidney and skeleton for several of the models are presented in Appendix C.

Dosimetry

The average radiation dose rate to bone can be calculated in a relatively straightforward manner, and Harley (Har74) derived the following formula in convenient units for environmental work:

$$D = 18.7\ C\bar{E},$$

where D is the dose rate in mrad/yr, C is the radionuclide concentration in pCi/g, and $\bar{E}$ is the energy absorbed per disintegration in MeV. Average doses calculated by this formula, normalized to 1 pCi/g of parent nuclide and adjusted for appropriate daughter equilibria and radon emanation, are summarized in Table 3. The dose rates per unit concentration of parent nuclide in bone decrease in the following order: $^{228}Ra > ^{226}Ra > ^{238}U$.

## Development of Limits for U

The committee believes that limits for natural U in drinking water should be based on chemical toxicity (which has been observed in man and quantified in animals), rather than on a hypothetical radiological toxicity in skeletal tissues (which has not been observed in either man or animals). The naturally occurring mixture of U isotopes, $^{234}U$, $^{235}U$ and $^{238}U$, has such a long collective rate of radioactive decay (half-life), and a correspondingly low specific activity, that its biological action is predominantly that of a nonradioactive element. That fact was considered to be compelling by the ICRP in its 1959 recommendations on permissible dose for internal radiation (ICRP59). Of all the radionuclides for which the ICRP recommended body burden or maximum permissible concentration limits, $^{238}U$ and its naturally occurring mixture with $^{234}U$ and $^{235}U$ were the only nuclides for which chemical toxicity was considered to be the limiting criterion.

There is as much or more information on the chemical toxicity of U as there is for any other metal (Tan51, Vo49, Vo53), and it has been the subject of more recent studies and reviews (Hod73a,b; Yu73, Hu73, Du75, Mo82, Wrenn, Morrow and Hursh, private communication to Union Carbide Corp., 1983-84). The quantitative relationships between U intake and kidney damage have been measured in several species over a large range of dosages of many soluble U compounds administered by various routes for, in some cases, extended periods of time. Only transient kidney dysfunction has been observed in patients given homeopathic injections of $UO_2(NO_3)_2$ (Hod 73b) or in U workers (Eas58, Wr75). Most recently, Moss and McCurdy (Mos82) reported increased $\beta_2$ microglobulin excretion in urine that could be correlated with U in well water at concentrations ranging from less than 0.5 to greater than 80 μg/liter;

however, the influence, if any, of other unspecified constituents of those waters, and total U intake, has not yet been taken into account, nor has the nor has the specific effect of U on $\beta_2$-microglobulin excretion been quantified.*

Proximal renal tubule cells are killed by high acute or lower chronic dosages of soluble U compounds administered by any route to experimental animals (Yu73, Du75). If the U dosage is not great enough to destroy a critical mass of kidney cells, the animal survives and the lost tubule cells are replaced. However, the new cells are not structurally identical to those lost, and presumably they are not functionally identical to them. It also appears that not all U-damaged tubule cells are replaced and that their nephrons are ultimately lost, as is shown by an increase in the proportion of connective tissue and a reduction in weight of chronically U-poisoned kidneys. Below some critical U concentration, mass law relationships favor the presence of U in glomerular filtrate as a soluble complex that does not react with the tubule cells; however, above that critical U concentration kidney damage might be expected to occur gradually. Although the renal reserve is great (only a fraction of nephrons are functional at any time), if U exposure is high for a long enough time, damage may accumulate to a level that compromises function.

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* U concentrations were increased as U concentrations in well water increased in the urine and hair of 133 persons who draw household water from wells with U concentrations ranging from less than 0.5 to greater than 80 $\mu$g/l (Mos82). The relationships are not altogether clear, e.g., linear regression equations predict significant U in both hair and urine of this group at zero concentration in the water. There are neither histories of, nor overt clinical signs of, kidney dysfunction even among those drinking the waters with the highest U concentration. No subtle changes of kidney function were revealed by clinical chemistry except an apparently elevated excretion of $\beta_2$-microglobulin (BMG, a low molecular weight protein, considered to be a sensitive indicator of certain renal tubular disorders). BMG excretion increased per unit of creatinine excretion with increasing U concentration in water, but BMG excretion was substantial, about 80 units BMG per unit of creatinine, even among those drinking waters with U concentrations less than 0.5 $\mu$g/l.

As was noted in the Introduction, bone cancer has been induced in experimental animals by injection or inhalation of soluble compounds of high specific activity U isotopes, $^{232}U$ or $^{233}U$ (Fin54, Ba183), but neither bone cancer nor marrow dysplasia have been reported among the many animals that have received compounds containing only the natural mixture of U isotopes. There are only two modern controlled animal experiments in which cancer was induced by natural U, but neither supports a conclusion that bone cancer would result from ingesting soluble U in drinking water or foods. In one experiment, lung cancer was produced in rats and dogs but not in monkeys following inhalation of large amounts of highly insoluble $UO_2$ continuously for 2 to 5 years (Le70, Le73): clearance of the $UO_2$ from the lungs was slow, and radiation doses of the order of 200 to 500 rad were accumulated in the lungs of those animals that developed lung tumors. In the other study, metallic U powder dispersed in lanolin was implanted in rats, either once into the marrow cavity of the femur (50 mg U) or in six monthly injections into the pleural cavity (50 mg U each). A small fraction of the U dissolved, as indicated by some acute mortality and late renal damage. Sarcomas containing U particles, reported to be of periosteal or connective tissue origin, developed at or adjacent to the U injection sites. The same or greater incidences of similar tumors at these same sites were seen in duplicate experiments with metallic Ni. The authors were unable to decide whether the local tumors induced by insoluble U were caused by its chemical or physical properties (Hue52). Sarcomatous tumors have also been induced in man by large, long-standing deposits of extravasated, insoluble, finely-divided $^{232}ThO_2$. (The primary alpha-emissions of $^{232}Th$ are comparable in energy to $^{238}U$.) (Sw67; Mays74). These observations are not relevant to ingestion of U in drinking water.

While the information concerning renal toxicity as a consequence of continuous intake of very small amounts of U is not as extensive or convincing as that obtained for higher levels of U intake, the committee believes that if any late effects will be associated with chronic ingestion of soluble U in drinking water, they are more likely to be chemical.

Our consensus metabolic model for U uses a GI absorption of 1.4%, single exponential elimination from the kidney with a half-time of 15 days, and a blood-to-kidney transfer of 11% to calculate the concentration in kidney as a function of time. In accordance with NAS guidance (NAS77, p. 804), the committee introduced a safety factor (called uncertainty factor by NAS) of 50 to ensure that individuals will be unlikely to experience permanent kidney damage from the ingestion of water containing uranium. The no-toxic-effects concentration limit in the kidney was taken as 1 μg U/g kidney. The committee considers irreversible kidney injury a non-stochastic effect with a threshold. In the past, 3 μg/g has been considered the approximate threshold (Sp73). As is described in Appendix C, we concluded daily intake should be limited to 187 μg U/day. For 1.7 l/day intake of water (NAS77, p. 11), the limiting concentration in water should be about 110 μg/l. A rounded value of 100 μg/l, is suggested, which is equivalent to 67 pCi/l of longlived alpha-emitting U isotopes, if the $^{238}U$ and $^{234}U$ isotopes are in radioactive equilibrium.

## Combining metabolism, dosimetry and risk estimates

The average per capita risk of bone sarcoma can be related to intake rate by the following formula, assuming linearity of risk and dose:

$$L_R = (IC)k ,$$

where: $L_R$ is the per capita lifetime average risk limit chosen for bone sarcoma per capita in the population of interest,

I is the per capita average fluid intake in liters/day,

C is the average concentration of Ra or U isotopes in water consumed (pCi/l),

k is the lifetime risk of bone sarcoma induction from 1 pCi/day intake over a lifetime. (From Ma84).

$k = 3 \times 10^{-7}$ sarcoma for $^{233,234,235,236,}$ or $^{238}$U

$k = 4.4 \times 10^{-6}$ sarcoma from $^{228}$Ra

$k = 3.2 \times 10^{-7}$ sarcoma from $^{224}$Ra

$k = 4.2 \times 10^{-6}$ bone sarcoma plus head carcinoma from $^{226}$Ra

The product IC is the daily intake of the nuclide (pCi).

As an example for natural U containing $^{234}$U and $^{238}$U in equilibrium, using d = 11 days, I = 1.7 l/day, $k = 3 \times 10^{-7}$ bone sarcomas/pCi/day per lifetime (Mays84), and $L_R = 10^{-4}$ (or about 10% of the average "natural" bone sarcoma rate in the U.S.), C is equivalent to 196 pCi/l of longlived U alpha activity. The toxicity limit chosen (67 pCi/l for equilibrium total alpha-activity of $^{234}$U and $^{238}$U) is equivalent for 1.7 l/day water intake to a lifetime linear stochastic risk of bone sarcoma induction of $3 \times 10^{-5}$; for 1 l/day average water intake the risk estimate is equivalent to $2 \times 10^{-5}$. We note that a somewhat lower lifetime (70-year) bone tumor risk of $1.6 \times 10^{-5}$ is calculated for intake of natural U in 1.7 l/day of drinking water containing 100 µg U/l, if we use the risk factors, bone surface cell dosimetry, and metabolic model and parameters for an equal activity mixture of $^{234}U$ and $^{238}U$ as recently recommended by ICRP (ICRP77a, 79), but substituting a GI absorption of 1.4% for the ICRP value of 5%.

The formula may be used to construct linear estimates of average risk. True expectations depend on the shape of the dose response, which if proportional to $D^2$ for example, would be near zero (see Mays84).

Table 4 lists the concentrations of $^{233,234,235,236}$ or $^{238}U$ and $^{228,226,}$ or $^{224}Ra$ in drinking water which, if ingested daily over a lifetime at a rate of 1 l/day, would imply lifetime risks of $10^{-4}$ and $10^{-5}$ of inducing bone sarcomas or bone sarcomas plus head carcinomas (in the case of $^{226}Ra$).

Note that the limit we suggest for soluble U in drinking water, based on renal toxicity, is less than the limit based on an "acceptable" risk of bone cancer ($L_D = 10^{-4}$) that was derived above. It should be noted also that the concentration of $^{238}U + {}^{234}U$ in radioactive equilibrium that can be calculated for water in the public domain using the new ICRP metabolic data, 50-year committed dose to bone surface cells, and based on risk of bone cancer (ICRP79) is 4770 µg U/l. That value is more than twice the old ICRP limit (ICRP64) based on chemical toxicity (1800 µg/l), even though the ICRP recently adopted a five-fold higher GI absorption fraction for U (5%). It would appear that, in the case of natural U, chemical toxicity is a more restrictive health effect criterion than risk of radiation-induced bone cancer, unless the cancer risk limit is set at an unrealistically low level.

## Summary

1. The metabolisms of U and Ra have been discussed with reference to limiting chemical toxicity effects on the kidney for U, and evaluating the risk of bone sarcoma induction, a stochastic effect, for Ra isotopes. In addition, estimates of potential risk of inducing bone sarcoma were made for U (Mays84), even though it is not established whether natural U can induce bone sarcomas.

2. We use the best estimates of each metabolic parameter to estimate intake, GI absorption, distribution, excretion and stochastic risk.

3. We use best estimates of the coefficients along with a safety factor to derive limits for chemical toxicity for U in water.

4. The committee concluded that a simple model of bone metabolism would suffice to evaluate the concentration of U or U isotopes in human bone under equilibrium conditions.

5. Equilibrium models for ingested nuclides that experience little placental discrimination are nearly independent of age. The number of days equivalent intake for the following nuclides in the skeleton at equilibrium are as follows:

| | | |
|---|---|---|
| $^{226}$Ra | 25 | days |
| $^{228}$Ra | 10 | days |
| $^{224}$Ra | 0.3 | day |
| Longlived U | 11 | days (range 1 to 35 days) |

6. A great deal of environmental and human and animal metabolic and toxicologic data underlies the Ra work. A lesser amount of human and animal

data are available for U. The epidemiological studies of $^{226}$Ra + $^{228}$Ra and $^{224}$Ra in man show that all these nuclides can induce bone sarcomas, that $^{226}$Ra can induce carcinoma of the soft tissues, no statistically significant increase in leukemias is noted (Spi83). To date, there are no data suggesting any radiological effects from ingested natural U in either animals or man.

7. The average dose rate to bone was summarized for these nuclides for a reference concentration of 1 pCi/g of 5000 g of mineralized tissue in Reference Man skeleton.

8. A recommendation was developed to limit chemical toxicity to the kidney from the ingestion of natural U in drinking water. The appropriate limit for soluble U in drinking water would be 100 μgU/l, using a metabolic model which estimates a higher kidney uptake of U than the new ICRP model (ICRP 79), limiting the U concentration to 1 μg U/g of kidney, and applying a safety factor of 50. This is equivalent to 67 pCi/l of longlived alpha-emitting natural U isotopes in their ordinary radioactive equilibrium.

9. The equivalent of 11 days intake was chosen as the equilibrium content of U in the adult human skeleton. The range of measured values is from 2 to 52, indicating that our estimate could be as much as a factor of 5 higher or lower than the extreme values.

10. The dose rate to bone from $^{226}$Ra was compared to that from $^{228}$Ra. For uniform distribution of the dose and for 90% retention of the $^{228}$Ra daughters, the dose rate to bone at equal intake rates of the two Ra nuclides is about the same. For practical purposes, it might be concluded that $^{226}$Ra and $^{228}$Ra are equally important in drinking water on an activity basis. $^{228}$Ra in the skeleton equals 10 days equivalent intake, but this is compensated by the greater average energy per disintegration delivered by its daughter series.

11. A lifetime risk of bone sarcoma induction equal to $10^{-4}$ would be conferred by lifetime consumption of 1.7 l of water per day containing 31, 32, and 174 pCi/l of $^{226}$Ra, $^{228}$Ra, and a combination of $^{234}$U, $^{235}$U, and $^{238}$U, respectively. A lifetime acceptable risk of cancer induction from 0.7 X $10^{-4}$ to 0.7 X $10^{-3}$ is suggested by ICRP (ICRP77a,b).

12. The committee concluded that a best estimate of dose and risk per capita should be made; uncertainties should be propagated; and conservative coefficients should not be used to evaluate the limiting cases for intake. The committee did not have the time and resources to analyze the data taking into account propagation of errors for the whole modeling process. This should be done.

13. The committee recommends a limit of 100 μg/l for natural U based on chemical toxicity to the kidney. This should be sufficiently low to make the likelihood of kidney damage to individuals remote. In addition, using a linear dose response model, the lifetime risk of bone sarcoma induction from natural U at that toxic limit for continuous intake would be between $10^{-5}$ and $10^{-4}$.

Recommendations

1. Use best estimates of metabolic parameters, realistic models, and propagate the uncertainties. The latter may require some research.

2. Further research is needed on GI absorption of U in animals (excluding rats), and on the toxicity and pharmacokinetics of U under conditions of chronic oral intake or its equivalent.

3. It is important to obtain more and better data for natural levels of U in water, diet, and human bone in different geographic areas. The distribution of U in the human body should be investigated more thoroughly, including soft tissues as well as the skeleton.

4. GI absorption of U should be inferred by measuring U intake and both fecal and urinary U excretion in man under controlled intake conditions.

5. Surveys of current water (and other fluid) intake are needed to better understand the average per capita intake of local drinking water.

6. Final limits for U in drinking water should not be set until the research identified above (at least items 3, 4, and 5) is complete. The research is reasonably short-term in nature and could be completed within a few years.

7. The interim $^{226}$Ra limits in water could be relaxed by a factor of at least 4, and still provide a very high degree of protection for individuals.

8. For interim guidance for U, 100 μg U/l of water was chosen as a reasonable value, based on considerations of kidney toxicity, with the application of a safety factor of 50 to 150.

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# Appendix A: Gastrointestinal absorption and accumulation of uranium in the body.

Patricia W. Durbin and McDonald E. Wrenn

## I. Animal Experiments

### A. Single administration of soluble U salts to adults

1. $^{233}UO_2(NO_3)_2$ nitrate in solution at pH 4 was given to 60-day-old rats by gavage at a dosage of 0.3 mg U/kg. Absorption was originally reported to be < 0.05% (Ham48, Du75). The original data were reanalyzed to take account of technical errors*. Absorption may have been as much as 0.35%, when recalculated by summing the U contents of tissues (except GI tract and skin) and also by comparing the U contents of skeleton, liver and kidneys with those of rats injected with U intramuscularly.

2. $UO_2F_2$ enriched with $^{233}U$ was dissolved in water and fed to dogs by mouth at a dosage of 0.7 mg U/kg (Fis60). Average absorption was reported to be 1.55% with a range for 7 dogs of 0.83 to 2.3%, based on assay of tissues and excreta. Only the final calculated result was originally reported, but the raw data were recently located and confirm the published mean value.

3. $^{232}UO_2(NO_3)_2$ or $^{233}UO_2(NO_3)_2$ in solution at pH 1.5 to 2 was given to adult rats by gavage at dosage levels of 2.3 µg $^{232}U$/kg or 4 mg $^{233}U$/kg (Su80a). Absorption at both dosages was 0.06%, determined by summing assayed tissues and urine to 7 days. The same results are obtained if the skeleton and liver U contents are compared with those of rats killed 7 days after an intramuscular injection of U (Harr81, Ham48, Du75).

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* Assay of $^{233}U$ was accomplished by dissolving ashed tissues, evaporating small aliquots on metal plates, and alpha counting. In this particular study, corrections for the self-absorption of the alpha particles, which is substantial in the aliquots of bone samples used, was inadvertently omitted. The minimum detectable amount had been assumed to be one-half of the background count regardless of the length of time of counting, and all samples yielding a net count less than one-half of the background count were disregarded. Recalculation involved applying the appropriate self-absorption corrections and using all data.

4. $^{233}UO_2(NO_3)_2$ in solution at pH 1.5 to 2 was given to adult rats by gavage at dosages of 5.1, 12.6, or 25.3 mg U/kg (Su83). At 7 days the skeletons contained 0.01, 0.01 and 0.02% of the gavaged U, respectively. Absorption of 0.044, 0.044 and 0.088%, respectively, can be calculated by comparison with U-injected rats (Harr81, Ham48, Du75).

5. $^{233}UO_2(NO_3)_2$ in 0.1 M $HNO_3$ was administered to adult Syrian hamsters by gavage at a dosage of 0.63 mg U/kg. Absorption was calculated to be 0.77% based on a comparison of the U contents of several body parts at 14 days with those of intravenously injected hamsters (Harr81).

6. $^{233}U$ in a chlorinated solution of 0.01 M $NaHCO_3$ was given by gavage to a fasting adult baboon at a dosage of 0.53 µg $^{233}U$/kg. At the same time, 0.42 µg $^{236}U$/kg was administered to the same baboon by intravenous injection. Absorption was estimated to be 1.2% based on comparison of the $^{233}U$ and $^{236}U$ contents of several body parts at 32 days (Lar84).

B. Continuous feeding of U salts to adult animals

1. Rats were fed for up to 2 years on pelleted diets containing 0.05 to 0.5% of $UO_2F_2$ or 0.5 to 2% of $UO_2(NO_3)_2$ (Ma53). If daily food intake was 5 g/100 g weight, daily U intakes ranged from 20 to 200 mg U/kg of uranyl fluoride and from 125 to 500 mg U/kg of $UO_2(NO_3)_2$. Animals were killed at 2 years, and the U content of their bones was determined, as shown in Table A-1.

If it is assumed that the ash of the rat skeleton is 4 g/100 g body weight, that the overall turnover rate of U in rat bone is 0.01 $day^{-1}$, and that the fraction of absorbed U deposited in bone is 0.2, then intestinal absorption is estimated as follows:

$$\text{Absorption (\%)} = \frac{\mu\text{g U/g bone ash} \times 40\text{ g bone ash/kg} \times 0.01\text{ day}^{-1} \times 100}{\text{mg U/kg/day intake} \times 1000\ \mu\text{g/mg} \times 0.2}$$

As shown in Table A-1, the range of estimated absorption is from 0.038 to 0.078%, and it appears to be independent of the concentration of U in the diet over the range investigated.

2. Adult male and female Sprague-Dawley rats (approx. body weight, 300 g) and male New Zealand rabbits (approx. body weight, 3.5 kg) were given drinking water ad lib containing 600 mg/liter of $UO_2(NO_3)_2$ (284 mg U/liter) for 90 days. The animals were killed, and the U content of bones and kidneys was measured. Intake of U was estimated to be 10 mg/day ($3.3 \times 10^4$ μg U/kg) for the rats and 60 mg U/day ($1.7 \times 10^4$ μg U/kg) for the rabbits. Absorption was calculated to be 0.035% for the rats and 0.28% for the rabbits, based on measured body content of U, the estimated daily intake, and the 9.74-day retention integral used for U in man (ICRP79, Tr83b). The value calculated for GI absorption of U in rats agrees reasonably well with values reported by others for single administration of U salts by gavage (Ham48, Su80a, Su83) and for continuous feeding of dry U salts mixed with the diet (Hav49, Ma53). See Table A-2.

3. $UO_2(NO_3)_2$ was fed to rats for 30 days at dietary levels from 0.5 to 12%. The mean lethal dosage at 30 days ($LD_{50/30}$) was a dietary level of 4% (972 mg U/kg/day based on an assumed food intake of 5 g/100 g weight/day). The $LD_{50/30}$ for intraperitoneally injected $UO_2(NO_3)_2$ in solution was 0.39 mg U/kg/day, from which it could be inferred that 0.39 x 100/972 = 0.04% of the dietary U had been absorbed (Hav49; Ma49).

C. Single administration of soluble U salts to neonates

1. $^{232}UO_2(NO_3)_2$ or $^{233}UO_2(NO_3)_2$ solutions at pH 1.5 to 2 were given by gavage to 2-day-old rats at dosages of 0.12 μg $^{232}U$/kg or 211 μg $^{233}U$/kg (Su80b). The skeletons contained 6.23 and 0.82% of the gavaged U at 7 days respectively, from which absorptions of 6.7 and 1.3%, respectively, were calculated.

2. $^{233}UO_2(NO_3)_2$ in solution at pH 1.5 to 2 was given by gavage to 1-day-old miniature swine at a dosage of 1.5 to 2 mg U/kg (Su82). Skeleton and tissues (except GI tract) were assayed at 12 days and contained 31.3 and 3.2% of the gavaged U, respectively. Absorption was estimated to be at least 34.5% from the body content, but a comparison with injected U would provide a more accurate result.

## II. Experimental administration of U solutions in man

1. $UO_2(NO_3)_2$ dissolved in water was drunk by an adult volunteer. The amount administered was 417 mg U, which for the Reference Man weight of 70 kg, was 6.7 mg U/kg (Bu58). An unknown amount of the U was lost by immediate vomiting, and the intestinal tract was cleared rapidly by the onset of diarrhea. In 7 days about 2.5 mg of U was recovered in urine. Comparison with urinary excretion of about 80% of intravenously injected U in 7 days (Ba48;Bu73) indicates that about 3.1 mg or 0.73% of the total ingested U had been absorbed.

2. $UO_2(NO_3)_2$ dissolved in Coca-Cola was drunk by four male hospital patients (56 to 78 years old) after an overnight fast (Bu73). The amount of U administered ranged from 0.08 to 0.17 mg U/kg. Uranium in urine was measured for one to two weeks, and mean cumulative excretion was 1.15% ± 1.26 at 7 days. Absorption, estimated by comparison with the U-injected hospital patients (50% of injected U excreted in urine in 7 days, Ba48), ranged from 0.3 to 3.4%, with a mean of 1.4% ± 1.4.

The results of the experimental studies of U absorption in animals and man are collected in Table A-2.

## III. Estimation of the absorption of U from environmental data

There is little endogenous fecal excretion of U after its injection in animals or people. Under equilibrium intake conditions, if the amount of U inhaled is small, daily excretion of U in urine approximates the U absorbed from food and drink. Thus, it should be possible to estimate intestinal absorption of U in man and animals from the U content of the alimentary intake, urine and feces (Bu73; Ad74; Wr77b).

### A. U in foods--dietary surveys

In regions where treated surface waters are used for cooking and drinking, foods appear to be the major source of environmentally acquired U (We67; No70; Ha72). The average concentration of U in rocks is about 4 ppm (Al60), and on the average, U is taken up by plants from the soil to the extent of $7.5 \times 10^{-5}$ g U/g fresh plant material (µg U/g dry soil) (Tr83a). Industrial fume and tobacco smoke may contribute small amounts of U; < 0.01 µg U/day from breathing city air and < 0.05 µg U/day from cigarette smoking (two packs) (Ha72; Lu70).

Average dietary intake of U has been calculated for three U.S. cities from the U content of a variety of foodstuffs purchased in those cities and the average annual consumptions of each of those foods obtained from a survey of foods purchased by households (USDA 1955, updated 1967, quoted by We67; Bu73). The per capita intake for household members in New York City and Chicago, cities from which urine samples were also obtained, was calculated to be 1.38 µg/day, with 1.35 µg/day derived from foods and 0.03 µg/day derived from 1 liter of water (We67).

Household food surveys do not take account of the differing intakes of men, women, and children. Based on caloric need (body size, activity, growth), the daily food intake for Reference Man (ICRP74) is 1.4 times that of women and 10-year-old children. All of the calculations in this review are for Reference Man, and we estimate that the intake of U by Reference Man in New York City and Chicago is 1.75 µg/day*.

A per capita dietary intake of 1 µg U/day was estimated for the U.K. in the same way (Ha72). Prepared foods, condiments, and dry tea and coffee were assayed for U in addition to foods as purchased. The U concentrations of prepared foods were all greater than those of the same foods as purchased. Table salt was found to contain 40 ppb of U, and 5 g of salt would add 0.2 µg U to the daily intake. Likewise, if U in tea and coffee were leached in preparation, that U would be added to the daily intake. The U in drinking water was not measured in the U.K. study, so it is incomplete in that respect. The data suggest that actual intake of U with foods and beverages is somewhat greater than intake calculated from surveys of foods purchased.

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* If, at the time of the food surveys, the average U.S. household was one adult male, one adult female and two children, their total caloric need would be about 3 x 1 unit plus 1.4 unit or 4.4 units of caloric need. The total U intake of the four-person household with a per capita intake of 1.38 µg U/day would be 4 x 1.38 = 5.52 µg U/day, and the consumption would be (5.52 µg U/day/household) /(4.4 caloric need units/household) = 1.25 µg U/day/unit of caloric need. If the food intake of Reference Man is 1.4 times greater than unit intake, the U intake of Reference Man in New York City or Chicago would be 1.4 x 1.25 µg U/day = 1.75 µg U/day.

Ranges of the measured U concentrations in the foods analyzed were also reported as follows: plant products (fruit, vegetables and grains, both raw and prepared) 0.3 to 30 ng U/g wet weight; and animal products (meat, fish, poultry, eggs, milk, both raw and prepared) 0.0005 to 4 ng U/g wet weight (Ha72). The U concentrations in the animal products were 1/10 or less of those in the plant products, which is not surprising considering the demonstrated discrimination by domestic animals against U in their diets (see Table A-2 and We67) and in secretion of milk (McC63; La82).

Replicates of daily diets of adults were measured for U in two Japanese cities, and the median U concentration was 0.042 μg U/g of food ash (No70). The ash content of the diet was estimated to be about 35 g/day, so the total intake of U in food by adults was calculated to be 1.5 μg U/day. Drinking water contributed 0.009 μg U/liter. When account is taken of the smaller size of adult Japanese (60 kg; Ta79) compared to U.S. males (Reference Man weighs 70 kg), that value for U intake is in good agreement with our calculated value of 1.75 μg U/day for the dietary U intake by Reference Man in the U.S.

### B. Excretion of U in urine

Urine samples were obtained from adults (probably all males and probably all laboratory employees) living in the New York City and Chicago areas, and the U concentrations were determined (We60; We67). The range of U concentration was the same for the two sample sets, and the results are combined in Table A-3. The distribution appears to be log normal, with a geometric mean of 0.097 μg U/liter of urine. Higher values have been reported from an industrial laboratory in a U-processing facility (Wi65) and quoted from an undocumented personal communication (M.H. Dean, quoted by Hu73). However, the urine U concentrations shown in Table A-3 were obtained by the same analysts who measured the U content of the U.S. foodstuffs, and the results of the two sets of measurements should be internally consistent.

### C. Calculation of U absorption

The daily urine volume of Reference Man is 1.4 liters, and for a U concentration in urine of 0.097 μg U/liter, urinary elimination of U would be 1.4 liter/day x 0.097 μg U/liter = 0.135 μg U, which is nearly equal to the amount per day at equilibrium. For a daily intake of 1.75 µg U by Reference Man, GI absorption would be (0.135 µg U/day) X 100/(1.75 µg U intake/day) = 7.7%.

D. Concurrent analyses of U in urine and local diet

Soils, stream and irrigation waters, local vegetables, composited human diets (food and drink), and human urine were obtained in and around three Japanese villages near U mines and mills, and from a remotely located control village. U was analyzed fluorometrically, and the minimum detectable amount was about 0.01 µg U. Only the results from Ten-no, the village with the highest level of environmental U, are considered here, because U was detectable in all the urine samples from that village. Daily intake of U in foods and water was about 9.2 µg for residents of Ten-no over 10 years old. Twenty-four-hour urine specimens were obtained on four occasions from about 25 men and women over the age of 10, for a total of 116 samples. The range of urinary U was 0.02 to 0.24 µg/24-hours, and the mean was 0.15 ± 0.043 µg U/person/day. The urinary U values appeared to be normally distributed about the mean and were independent of sex, age, or time of sampling (Ya68, Mas71a,b,c,d).

An average apparent U absorption, 0.15 ± 0.043 µg U in 24-hr urine/9.2 µg U intake/day = 1.6% ± 0.5, can be calculated from these data. Data from the control and the two other U mining villages yielded estimates of U absorption less than 1.6%; and they are skewed to low values by the large numbers of urine samples with less than the minimum detectable amount of U, to which zero values were applied. The strengths of this body of data are the large number of urine samples analyzed, the breadth of sampling, and the concurrent analysis of U in local diets. Its weaknesses are the insensitivity of the analytical method used for U and the poor recoveries of U from food and soil samples.

E. Estimation of U intake from fecal U excretion

1. Control subjects in a study of the differential metabolism of $^{230}$Th and $^{234}$U and $^{238}$U inhaled in U ore dust included three retired U mill workers (4 to 14 years since last employment as U ore crushermen), and three volunteers who lived in U milling communities but had no U work history. Two consecutive 24-hr urine and fecal collections were obtained and analyzed for $^{234}$U and $^{238}$U. The data for $^{238}$U, which are supported by the results for $^{234}$U, are shown in Table

A-4 (Fish83). Apparent U absorption can be calculated assuming that the sum of daily urinary and fecal U excretion is equal to the alimentary intake. It should be noted that U absorption will be underestimated if insoluble U compounds are being inhaled, or if, in the case of the retirees, insoluble U is still being cleared mechanically from the lungs by way of the GI tract. Conversely, U absorption will be overestimated if soluble U is being inhaled, or if, in the case of the retirees, U ore dust in the pulmonary tract is still being solubilized and absorbed into the body.

The apparent total intakes of U of these individuals ranged from 11 to 18 µg U/day for the controls and from 5.3 to 71 µg U/day for the retirees. Although large compared to U intakes estimated for city dwellers (We67; Ha72), the U intakes of these individuals are not unreasonable, because U in potable waters and locally grown foods tends to be higher in U mining and milling communities. The mean U absorption calculated for the three controls (0.82%, range 0.6 to 1%) was not significantly different from that calculated for the retired U workers (0.94%, range 0.55 to 1.6%) and the two groups can be combined. This body of data suggests that at an apparent mean daily intake of 24 µg U/day (0.34 µg U/kg for 70 kg Reference Man), the GI absorption of U is 0.76% (range 0.4 to 1.6%).

2. In the city of Ahmedabad (Gujarat State of India) about 2 million people consume well water with U concentrations up to 22.4 µg U per liter. Complete 24-hour urine and fecal specimens were collected from an individual once in summer and once in winter and analyzed for U content. U absorption can be estimated assuming that it is not lost in sweat. The estimated U absorption for the summer sample, when about 3 liters of water was consumed daily, was urinary U/(urinary U + fecal U) = 2.25 µg U/(2.25 + 56.9) µg U = 3.8%. For the winter sample, when water intake was about 1 liter/day, U absorption was 0.18 µg U/(0.18 + 31.2) µg U = 0.57%. The mean of the two estimates is 2.2% U absorbed (So80). The high U content of the prepared vegetarian diet, about 20 µg U/kg, is not surprising considering the high U content of the local water. For an individual with a body size similar to Japanese Reference Man (Ta79), the mean daily intake is 0.76 µg U/kg.

The results of the four estimates of U absorption in man obtained from measurement of environmental U in ingesta and excreta are collected in Table A-5.

IV. Estimation of U absorption by comparison with Ca (Observed Ratio method)

The observed ratio (OR) was originally defined to predict the behavior of a metal (M), such as $^{90}Sr$, with respect to its well-studied essential analogue, calcium (Co60), as follows:

$$OR(body/diet) = M(body)/Ca(body) \div M(diet)/Ca(diet).$$

The U in water, plant products, and mammalian tissues is presumed to be in the stable +6 state as $UO_2^{+2}$ (Do49; Lat52; Al60; Ru66; Ya68). $UO_2^{+2}$ ion can (1) enter all domains of bone water, e.g., canaliculi, (2) participate in ion exchange processes at mineralized bone surfaces both by replacing Ca and subsequently being replaced by Ca, and (3) when accumulated at growth sites, be buried by formation of new bone (Ne51; Tan51; Ne58; Ro68; Pr82; Ste80).

When U is administered to animals or to man, most of the U present in the body after the initial phase of plasma clearance and urinary excretion is in the skeleton (Yu73; Du75; Ste80), and the major fraction of U acquired by people from the environment is in the skeleton (see references in Tables B-1 and B-2). The environmentally acquired U present in the human skeleton appears to be fairly uniformly distributed in bone mineral; however, there may be some accumulation of U on bone surfaces in adult life (Ha71). Although the chemistry and kinetics of Ca and $UO_2^{++}$ in bone are not exactly alike, $UO_2^{+2}$ is similar enough to Ca to warrant application of the OR method to obtain an independent estimate of the absorption of environmentally acquired U.

The OR(body/diet) is the resultant of several processes in which the foreign metal is not handled as efficiently as Ca, i.e., there is discrimination against the foreign metal. The most important discriminations against $UO_2^{++}$ occur in the kidneys, where Ca is efficiently reabsorbed; in the intestine, where to the degree that metabolic needs are met, Ca is actively absorbed; and in the skeleton, where $UO_2^{++}$ is limited to the hydration shell of bone mineral crystals (Ne58). The following relationships apply:

OR(body/diet) = DF(absorption) x DF(excretion),

DF(absorption) = fraction of metal absorbed/fraction of Ca absorbed,

DF(excretion) = fraction of metal retained/fraction of Ca retained.

Table A-6 contains the data required to calculate OR(body/diet) for U and the discrimination factors needed to estimate intestinal absorption of U.

For U, OR(body/diet) = $(38 \times 10^{-6} g/1000\ g)/(1.75 \times 10^{-6} g/day/1.1\ g/day)$
= 0.024.

Not surprisingly, that low value OR for U is the same as has been calculated for Ra (UN77, see text).

DF(excretion to 10 days) = 20/73 = 0.27.

U absorption (%) = OR(body/diet)/DF(excretion to 10 days) x % Ca absorbed
= 0.024/0.27 x 34 = 3.0%.

The intestinal absorption of U calculated from the "best" values for body U using the OR method is thus 3.%, less than one-half the value of 7.7% calculated from U.S. urine and diet data.

If the procedure outlined above is reversed, and it is assumed that the U analyses of U.S. diet and urine are the more reliable, the calculated U body content is about 100 μg, providing support for the higher reported values for U in bone.

## V. U absorption and intake level

The mean U absorption determined in 13 experiments with rats (Table A-2) was 0.078% ± 0.083, and if the least reliable result is omitted (Ham48), the mean for 12 rat studies is 0.055% ± 0.016. The mean U absorption for 6 experimental studies of U absorption in man and animals other than rats (Table A-2) combined with the four estimates of U absorption obtained from environmental data (Table A-5) is 1.86% ± 2.16. If the least reliable result, that based on U.S. dietary survey data (We60, We67) is omitted, the mean of nine experimental results is 1.2% ± 0.57. The difference between the mean U absorption determined for rats and that for all other animals is statistically significant (t-test, $p < 0.01$), regardless of whether the least reliable point in each group is omitted, indicating that the rats constitute a separate population, which should not be used in predicting U absorption for man.

Linear regression analysis was used to test the two data sets (rats and all other animals) for dependence of U absorption on U intake level. The regression equations and correlation coefficients of linear, semilogarithmic and logarithmic fits are shown in Table A-7. The logarithmic fit, selected for display in Fig. A-1 because all the data can be shown together, also provides the best fit to each set of data as indicated by the correlation coefficients. The slopes of the fitted lines for the two sets of U absorption data are probably not different from each other. An inverse dependence of the GI absorption of U on U intake is suggested by these data, but is not proven because the scatter of the data does not permit rejection of zero slope for either regression equation. All of the available data for man and animals other than rats could be fitted by a single line. The large displacement from the fitted line of the environmental point based on U.S. dietary survey data suggests that additional studies are needed to determine the shape of the U absorption curve in the region of very low intake.

Calcium is absorbed from the intestine by a specific active transport mechanism. For Ca intakes below that required to meet metabolic needs, fractional absorption is inversely dependent on intake, and thereafter fractional Ca absorption is nearly constant. GI absorption of the other alkaline earths also depends on dietary Ca levels, and they are presumed to be absorbed to some degree via the Ca transport system. The relationships between U absorption and levels of dietary Ca have not been studied, but the divalency and small ionic radius of the uranyl ion (Lat52, Sh76) may allow $UO_2^{+2}$ to be absorbed via the Ca transport system.

Some dependence of U absorption on intake is anticipated from chemical considerations. At very low levels of U intake, absorption should be maximal because of (a) the availability of natural complexing agents, e.g., citrate, to stabilize $UO_2^{+2}$ against reduction and precipitation and facilitate absorption, and (b) the thermodynamically favorable (mass law) condition of a low U concentration. Exhaustion of complexing agents in the intestinal contents and formation of insoluble diuranates and phosphates in the neutral to alkaline intestinal contents (Lat52) would act to reduce U absorption at high intake levels.

It is obvious that the point giving 7.7% absorption based on dietary survey data and fluorometric analysis of urine greatly influences the predicted U absorption at environmental levels. Curve 1 of Figure A-1, which includes that point and the other experimental data for animals other than rats, predicts 3.4% of U absorbed at an intake of $2 \times 10^{-2}$ μg U/kg/day. Elimination of that point from the data set yields a predicted absorption of 2.1% of U at that intake level (curve 2). In the absence of the resolution of some of the uncertainties, we consider that for the purposes of this report, and over the range of U intake of concern in setting drinking water standards (up to 2 μg/kg/day, 1.4 l/day of water containing about 35 pCi U/l consumed by Reference Man), it is reasonable to use a rounded-off value of the mean that was calculated for man and animals other than rats, U absorption = 2%. Our best estimate of absorption at 1 μg/kg/day U intake is 1.4%. None of the available experimental or environmental data support a fractional U absorption greater than about 5%, even at intakes of the order of 1 to 2 μg/day for Reference Man. A higher value for U absorption (about 20%) based on dietary U data from the U.K. (Ha72) and unpublished analyses of U in urine (M.H. Dean, quoted by Bu73) seems unlikely on physiological grounds, because it approaches the fractional absorption of Ca and Sr (ICRP73).

## Appendix B: Distribution of U in the body

Patricia W. Durbin and McDonald E. Wrenn

Distribution of U has been studied after administration to animals by injection, feeding or inhalation and to people by injection (see references in Hu73, Yu73, Du75, and Ma53, Sto51, Ber57, Li81, Harr81, Ste80, Su80ab, Su82, Su83, Pr82). Bone is the major site of U accumulation in all the mammals studied, whether the U exposure was a single or prolonged experimental administration or by intake from the environment. The relationship between the U content of the human body and the levels of U in the environment (food, water, and air) is not clearly established (Be64, Hu73, Sp73, No62); however, continuous U feeding or inhalation in animals demonstrate that the amount of U in bone, and to a lesser degree in soft tissues, is directly proportional to the U exposure level. Equilibrium U concentrations were rapidly established in all the tissues of growing animals and in the soft tissues of animals that were skeletally mature at the start of exposure (Ma53, Sto51, Le70). From the animal experiments, it can be inferred that nearly constant U concentrations should be found in people exposed to a constant level of environmental U, that U turnover in soft tissues is fairly rapid, and that turnover of some fraction of U in bone is slow.

The chief difference between the distributions of administered U in people (Ber57) and larger animals (dog, Tan51; monkey, Le70; baboon, Li81) and those in small animals (mainly rodents; Yu73, Du75) is that for the first few days to weeks a larger fraction of the U is present in the soft tissues of the people and larger animals. For that reason, only the U studies in the large animals appear to be suitable for assessing the validity of the measurements of the distribution of environmental U in man. Urinary excretion of U is rapid but not highly efficient, and the significant U content of the soft tissues must be taken into account in the development of a model that accurately describes the metabolism of environmentally acquired U.

### I. Bone

Several sensitive analytical techniques have been applied in the U.S. and elsewhere to measure the level of U in the skeletons of persons with no known occupational U exposure, and the results are collected in Table B-1. [Note:

Other reports have appeared of "normal" U in the human skeleton and tissues with results that are ten or more times higher than those shown in Table B-1. However, the analytical methods used were not as specific for U nor as sensitive as those used in the studies tabulated, and more importantly, most of those other measurements were done in laboratories with high potential for contamination of environmental samples (Ca75, Bu58, Qu58; see also references in Hu73). Consequently, those results have not been included.] Nine sets of analyses for U in human bone specimens from several U.S. locations have been reported. The number of individual skeletons sampled is not available in all cases, nor can the associated alimentary and air intakes of U be assessed. The reported average values (recalculated to a common basis of ng U/g bone ash) range from 2.3 to 61.6 μg U in the skeleton of Reference Man. The best value to use at the present time appears to be the arithmetic mean (± S.D.) of the nine studies, 24.9 ± 22 μg U, in the 5000 g of bone (2800 g of bone ash) of Reference Man skeleton. That choice is supported by the mean of the five skeletal U values reported for other places, 29.2 ± 20.2 μg U in the Reference Man skeleton.

## II. Soft Tissues

The amount and distribution of U normally present in human soft tissues is even more difficult to assess from the available information. There are fewer reported analyses (see Table B-2); some important tissues have not been analyzed, (skin, GI tract); some tissues have been analyzed by only one investigator in one location; and fat and skeletal muscle, which appear from the injection experiments to contain most of the U in soft tissues, have been analyzed only once in samples obtained outside the U.S. Consequently, it has been necessary to make some judgments, based on the results from the human U injection cases and from experiments in large animals.

Lung: The reported U concentrations in normal lung are 8 to 10 times higher than would be expected from the human and animal injection studies (Ber57, Tan51, Ste80, Li81). The unexpectedly high lung U content, and in addition the very high U concentrations in the tracheobronchial lymph nodes (TBLN), indicate that much of the U in the lungs (we shall assume 85%) was acquired by inhalation of insoluble material (Sto51, Le70).

Fat: Only one analyst reported on U in human fatty tissue, and then only for two specimens (whether those were from different individuals was not stated). That U concentration, 0.6 ng U/g wet tissue, is 1.5 times greater than the values reported by that and other analysts for spleen and kidneys, and two to three times greater than the reported normal U concentrations in liver, muscle and heart. In the human U-injection study, the concentration in fat ranked seventh or eighth of the eight tissues that contributed most to the total U in soft tissues (apart from kidney) (Ber57). The U concentration in fat was not more than two times that of skeletal muscle. The U concentrations in fat of one baboon 4 days after U injection was low; it was 2.9 times that of muscle, and less than one-tenth that of liver and spleen (Li81). In the other animal studies, U concentration in fat was not reported, but bone, urine and other soft tissues approached 100% of the administered U, so there was little U left over that could have been present in fat. On balance, it does not seem likely that fat contains 50% of the total U normally present in human soft tissues. As an alternative, we have elected to use the mean U concentration in heart and skeletal muscle as representative of the 32 kg of soft tissues and fat otherwise unaccounted for, and we estimate the U content of all of those tissues of Reference Man to be 5.6 μg.

Blood: The mean of the three reported sets of U assays of human whole blood, 0.46 ng U/ml, would lead to a calculated total of 2.4 μg of U in the blood volume of Reference Man. At an intake of 1.75 μg U/day, and an absorption of 2%, the blood volume would contain all of the U absorbed in 69 days. Considering the rapid efflux of parenterally administered U from plasma to extracellular fluid, bone and urine (Tan51, Ber57, Ro68, Li81, Ste80), the values reported for U in blood seem unduly high, unless a significant fraction of blood uranium is associated with cells. Analysis of U in separated normal human plasma and red cells (Lu70) and of the separated blood constituents in U-injected baboons (Li81) indicates that within a few hours after intake most of the U present in blood is associated with cells. The value assigned to blood in Table B-3 has been calculated to include the reported approximate partitioning of blood U between the plasma and cells of the baboon, assuming a venous hematocrit of 0.45.

Total soft tissue U: The value we have selected to represent soft tissue U, 13 μg, therefore excludes the TBLN and 85% of the measured lung U content, uses the mean reported U concentrations in heart and skeletal muscle as representative of the non-parenchymous soft tissues, and excludes the one-half of the blood volume that remains in organs obtained in a routine autopsy.

The "best" value for the normal U content of Reference Man, 38 μg U, of which 66% is in the skeleton, is in reasonably good agreement with the distribution of U experimentally administered to animals (Du75, Ste50) and human subjects (Ber57).

## Appendix C: Metabolic Models for Uptake of Uranium in the Human Kidney and Skeleton

### Kidney

For a two-compartment model of kidney, as proposed by ICRP (ICRP79), the equilibrium concentration at constant daily intake (I) is as follows:

$$\frac{A_\infty}{Im} = \frac{f_1}{m(\ln 2)} \left[f_{21}T_1 + f_{22}T_2\right]$$

where

$A_\infty$ = amount of U in the kidney at equilibrium

$m$ = kidney mass (310 g)

$f_1$ = fractional GI absorption from GI tract to blood

$f_{21}$ = fractional transfer from blood to kidney compartment 1

$f_{22}$ = fractional transfer from blood to kidney compartment 2

$T_{1(2)}$ = half-times in kidney compartments 1 or 2

$I$ = daily intake (μg).

Then

$$\frac{C_\infty}{I} = \frac{A_\infty}{Im}$$

is the concentration (C) in μg U per g kidney per μg/day intake. This value is listed in Table C-1 using the most recent ICRP model for U (ICRP79), and the continuous intake model used earlier by ICRP (ICRP59) and discussed by Spoor and Hursh (Sp73). In the table, $f_1$ is varied from 1 to 10% to show the effect of GI absorption on eventual kidney content. The measurements made after intravenous administration of U to comatose patients have been summarized (Bu73), and based on these, a blood-to-kidney transfer factor of 11% is assumed, with a 15 day half-time in the kidney.

If $C_L$ is the limiting concentration in kidney, and S is the additional safety factor desired, then the limiting daily intake ($I_L$) can be derived as

$$I_L = \frac{(C_L/S)}{(A_w/m)} .$$

$C_L$ has been generally accepted (Sp73) as about 3 μg/g kidney, but more recent data in dogs (Mo82) suggest that a value of 0.6 μg/g is below the injury threshold in man. We will use 1 μg/g to derive a limit; $A_w/m$ is taken from the last column in Table C-1. For the same value of $f_1$, the Spoor and Hursh model gives nearly the same equilibrium uptake in kidney as the ICRP model, exceeding that estimate by only 15%. We will use the simpler Spoor and Hursh model here.

We have used 1.4% as the best available GI absorption estimate for man at environmental levels of U intake and introduced a safety factor (S) of 50. The size of the safety factor depends on our certainty about the potential toxicity of U in man: safety factors have been called uncertainty factors by NAS (NAS77). We have some information about U uptake, absorption and metabolism in man, and a wealth of animal toxicology data, even though pieces of information that would improve the reliability of our estimates are missing.

The committee believes that based on the NAS definition, U should be assigned an uncertainty (safety) factor between 10 and 100 (NAS77, p. 804). We have chosen 50 as a factor that should provide a high margin of safety. One might argue that we have actually introduced a safety factor of 150, because we have also chosen a more conservative estimate (1 μg/g) for the limiting concentration of U in the kidney than was used in the past (3 μg/g).

We note that ICRP (ICRP64) derived a limit for occupational exposure to U in water ($MPC_w$) of $2 \times 10^4$ pCi/l, equivalent to $6.1 \times 10^4$ μg/l for 750 ml/day intake of water (Hu73, p. 253), or an intake by mouth of $4.6 \times 10^4$ μg/day. For individuals in the general population, the ICRP recommendations were equivalent to 1800 μg/l in water (Hu73, p. 263). Inasmuch as the ICRP had inadvertently underestimated GI absorption by about a factor of 10, this would be equivalent to 180 μg/l for 1% GI absorption.

We obtain $A_w/m = 1 \times 10^{-4}$ and $I_L = (1/50)/10.7 \times 10^{-5}$, or 200 μg/day. For 1.7 l of water intake/day for a reference individual (NAS77) and a safety factor of 50, this is equivalent to 120 μg/l. The committee recommends 100 μg/l (rounded off) as the intake limit for U in drinking water. It is designed to limit toxic effects in the kidney from chronic U intake. This U concentration has a radioactivity of 67 pCi/l of alpha-particles, if the naturally-occurring U isotopes are present in their normal relative abundances.

Skeleton

The equilibrium level of U in the human skeleton resulting from a constant and continuous daily intake at 1 unit per day in food and/or water is given by $A_w/I$ in the same formula as for kidney uptake, but with different numerical values of the coefficients. The general form of the ICRP model for U in bone will be retained here, namely two compartments for bone, one with a short half time (we choose 300 d instead of the 20 d chosen by ICRP), and the other with a long half time (5000 d, the same as the value chosen by ICRP).

Use of the coefficients for U uptake and fractional transfer from blood chosen by the ICRP for workers ($f_1 = 0.05$, $f_{21} = 0.2$, $f_{22} = 0.23$) leads to equivalent days accumulation in skeleton of ~ 8.3 days. Changing the half time for short-term bone to 300 d leads to an estimate of 12.5 days equivalent uptake. This appears to be on the low side of the various estimates of skeletal accumulation (see Appendix A), even though the value for $f_1$ used in the model is also higher than our best estimate of GI uptake (1.4%).

One may vary the coefficients in the model within ranges which are reasonable and consistent with animal experiments and human data (Du75). The animal data all point to a blood to skeleton transfer from 10 to 20% ($f_{21} + f_{22}$). For example if we assume $f_1 = 0.01$, $f_{21} + f_{22} = 0.2$, blood to short-term bone transfer of 0.18 and to long-term bone, 0.02, the model predicts 1.5 days equivalent accumulation, which would correspond to levels roughly equivalent to those reported by Fisenne (Fi80). At the other extreme using $f = 0.05$, and equal partitioning between long- and short-term bone, the equation predicts 26.5 days equivalent intake in bone.

We believe that estimating days equivalent intake from various analyses of human bone samples, coupled with dietary measurements of a general nature, produces an estimate of days equivalent intake with a large potential error. If we use our best estimate of human GI absorption the model predicts a skeletal content at the lower range of the observed values.

To estimate potential radiation risk to skeleton we will assume an equilibrium with 11 days equivalent intake in the skeleton as our best estimate (see Appendix A for the derivation).

Higher estimates are not excluded. For example, the ICRP metabolic data for Reference Man predicts 59 μg U in skeleton from a daily intake of 1.9 μgU/day or

31 days equivalent intake, while the metabolic model ICRP adopted gives 8 days (ICRP79). On grounds of comparative chemistry and skeletal biochemistry, $UO_2^{+2}$ should be less tightly held in bone than $Ra^{+2}$, and one would predict a shorter net residence time and smaller value for days equivalent intake for U than for Ra.

Cothern (Co83b) has obtained 33 using the ICRP model modified for 20% GI absorption. The 11-day equivalent accumulation in skeleton (range 2 to 33 days) cannot be said to differ significantly from the 8-day ICRP prediction, using the committee's consensus for the best values of the metabolic parameters. In order to determine the reasons for the difference, skeletal U burden data are required in a population with U intakes that have been well-characterized over decades. Data are available for $^{226}Ra$; however, for practical reasons, U data are not obtainable directly from living man, but must be inferred from bone samples obtained at autopsy.

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TABLE 1. Body $^{226}$Ra contents and daily $^{226}$Ra intakes (inferred from fecal excretion) of adult and adolescent human males. Their $^{226}$Ra was accumulated entirely from environmental sources (food, water, air). Data of Stehney and Lucas (St56).

| Group | No. of subjects | Mean age (years) | No. of fecal samples | Excretion per day*: Dry feces (gm) | Excretion per day*: Radium ($10^{-12}$gm) | Ratio of body Ra to fecal Ra |
|---|---|---|---|---|---|---|
| Adult Control | 1 | 29 | 3 | 26.6 ± 0.1 | 1.7 ± 0.4 | 24 |
| Stateville | 11 | 44 | 19 | 24.1 ± 3.8 | 12.6 ± 2.4 | 17** |
| Chicago boys | 7 | 17 | 26 | 24.7 ± 1.1 | 1.6 ± 0.1 | 22 |
| Lockport boys | 8 | 17 | 24 | 37.3 ± 3.2 | 8.2 ± 0.6 | 45 |

* Errors shown are the standard deviation of the mean for the group.

** Ratio for $^{226}$Ra retained by 11 subjects during a 19.7-year average period at Stateville.

Table 2. Days equivalent intake of radium isotopes in bone.

| Nuclide | Days Equivalent Intake | Distribution In Bone |
|---|---|---|
| Ra-226 | 25 | volume |
| Ra-228 | 10 | volume |
| Ra-224 | 0.3 | surface |

TABLE 3

Average skeletal dose rates (mrad/yr) from constant concentrations of naturally-occurring uranium and radium radionuclides, assuming a constant concentration of 1 pCi/g of parent nuclide and a uniform distribution.

| Nuclide | mrad/yr |
|---|---|
| $^{238}$U | 78 |
| $^{234}$U | 89 |
| $^{228}$Ra (+ dtrs. adjusted for 90% $^{220}$Rn retention) | 560 |
| $^{226}$Ra (+ dtrs. adjusted for 33% retention of $^{222}$Rn in bone, excluding $^{210}$Po) | 207 |
| $^{226}$Ra (+ dtrs. adjusted for 33% retention of $^{222}$Rn in bone, including $^{210}$Po) | 240 |

From Wr77b. If $^{238}$U and $^{234}$U are in equilibrium, add the two doses together. Alpha dose rates at bone/surface interfaces will generally be less than half the average dose rates in mineral bone.

Table 4. Computed limiting concentrations (pCi/l) of longlived U, $^{228}$Ra, $^{226}$Ra, and $^{224}$Ra in water for given lifetime risks ($L_R$) of bone sarcoma (plus head carcinoma for $^{226}$Ra) based on drinking water intake of 1 l/day

| Lifetime Risk ($L_R$) | Computed Limiting Concentration (pCi/l) | | | |
|---|---|---|---|---|
| | $^{233,234,236,236 \text{ or } 238}$U | $^{228}$Ra | $^{226}$Ra | $^{224}$Ra |
| $10^{-4}$ | 330 | 23 | 24 | 310 |
| $10^{-5}$ | 33 | 2.3 | 2.4 | 31 |

Table A-1. Estimated gastrointestinal absorption of U continuously fed to rats as dry salts mixed with diet. Data of Maynard et al. (Ma53). Food intake is assumed to be 5 g/100 g weight/day.

| Compound | Dietary U (g/100 g) | U intake (mg U/kg/day) | U in femur (µg U/g ash) | Calculated absorption (%) |
|---|---|---|---|---|
| $UO_2F_2$ | 0.01 | 3.8 | control | 0.052 |
| (0.77 U by | 0.05 | 20 | 5.2 | 0.059 |
| weight) | 0.25 | 96 | 28.4 | 0.059 |
|  | 0.50 | 195 | 58.2 | 0.06 |
| $UO_2(NO_3)_2\ 6H_2O$ | 0.50 | 118 | 22.5 | 0.038 |
| (0.47 U by | 2.0 | 472 | 184 | 0.078 |
| weight) |  |  |  |  |

Table A-2. Experimental determinations of gastrointestinal absorption of U.

| | Species | U Absorption (%) | U intake (µg U/kg/day) | Comments | Reference |
|---|---|---|---|---|---|
| **Adult animals** | | | | | |
| 1 | hamster | 0.77 | $6.3 \times 10^2$ | $^{233}UO_2(NO_3)_2$, pH 1 | Harr81 |
| 2 | rabbit | 0.28 | $17 \times 10^3$ | $UO_2(NO_3)_2$ in water | Tr83b |
| 3 | dog | 1.55 | $7 \times 10^2$ | $^{233}UO_2F_2$ in water | Fis60 |
| 4 | baboon | 1.2 | $5 \times 10^{-1}$ | $^{233}UO_2(NO_3)_2$ in 0.1 M $NaHCO_3$, pH 8.1 | Lar84 |
| 5a | man | 0.73 | $6.7 \times 10^3$ | $UO_2(NO_3)_2$ in water | Bu58 |
| b | man | 1.4 | $1.32 \times 10^2$ | $UO_2(NO_3)_2$ in cola drink | Bu69 |
| 6a | rat | 0.35 | $3 \times 10^2$ | $^{233}UO_2(NO_3)_2$, pH 4 | Ham49 |
| b | rat | 0.06 | 2.3 | $^{232}UO_2(NO_3)_2$, pH 2 | Su80a |
| | | 0.06 | $4 \times 10^3$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su80a |
| 6c | rat | 0.044 | $5.1 \times 10^3$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su83 |
| | | 0.044 | $1.3 \times 10^4$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su83 |
| | | 0.088 | $2.5 \times 10^4$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su83 |
| 6d | rat | 0.035 | $3.3 \times 10^4$ | $UO_2(NO_3)_2$ in water | Tr83b |
| 6e | rat | 0.052 | $2 \times 10^4$ | $UO_2F_2$ in diet, 0.05% | Ma53 |
| | | 0.059 | $9.6 \times 10^4$ | $UO_2F_2$ in diet, 0.25% | Ma53 |
| | | 0.06 | $2 \times 10^5$ | $UO_2F_2$ in diet, 0.50% | Ma53 |
| | | 0.038 | $1.2 \times 10^5$ | $UO_2(NO_3)_2$ in diet, 0.50% | Ma53 |
| | | 0.078 | $4.7 \times 10^5$ | $UO_2(NO_3)_2$ in diet, 2.0% | Ma53 |
| | | 0.04 | $9.7 \times 10^5$ | $UO_2(NO_3)_2$ in diet, 4.0% $LD_{50/30}$ days | Hav49 |
| **Neonatal animals (one to two days old)** | | | | | |
| 7a | rat | 6.7 | 0.12 | $^{232}UO_2(NO_3)_2$, pH 2 | Su80b |
| | | 1.3 | $2.1 \times 10^2$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su80b |
| 8 | swine | 34.5 | $2 \times 10^3$ | $^{233}UO_2(NO_3)_2$, pH 2 | Su82 |

Table A-3. Measurements of U in human urine. Data of Welford *et al.* (We60) and Welford and Baird (We67).

| U Concentration (μg U/liter) | Number of Samples | | |
|---|---|---|---|
| | Chicago | New York | Total |
| < 0.050 | 1 | 3 | 4 |
| 0.051 - 0.075 | 4 | 4 | 8 |
| 0.076 - 0.10 | 4 | 6 | 10 |
| 0.101 - 0.125 | 0 | 3 | 3 |
| 0.126 - 0.15 | 0 | 4 | 4 |
| 0.151 - 0.175 | 1 | 2 | 3 |
| 0.175 - 0.20 | 1 | 1 | 2 |
| 0.201 - 0.225 | 0 | 1 | 1 |
| 0.226 - 0.25 | 0 | 0 | 0 |
| 0.251 - 0.275 | 0 | 0 | 0 |
| 0.276 - 0.30 | 0 | 2 | 2 |

Total of 37 samples: arithmetic mean, 0.098 ± 0.067 μg U/liter; modal value = 0.088 μg U/liter (between 0.076 and 0.101 μg U/liter); geometric mean = 0.097 μg U/liter.

Table A-4. Gastrointestinal absorption of U calculated from urine and fecal data reported by Fisher *et al.* (Fish83).

| Subject | No. days | Apparent intake[a,b] (µg) | Absorption (%)[c,d] | |
|---|---|---|---|---|
| | | | $^{234}$U | $^{238}$U |
| C1 | 1 | 18 | 0.43 | 0.64 |
| C2 | 2 | 12 | 0.96 | 0.98 |
| C3 | 2 | 11 | 0.39 | 0.85 |
| R1 | 2 | 24 | 0.58 | 0.55 |
| R2 | 2 | 5.3 | 1.11 | 1.62 |
| R4 | 2 | 71 | 0.40 | 0.55 |
| Average | | 24 | 0.65 | 0.87 |

[a] Apparent intake = urinary U/day + fecal U/day. It is given only for $^{238}$U in µg.

[b] The daily intake of U is inferred from the specific disintegration rate of $^{238}$U: 0.742 dpm/µg U.

[c] The data were reported as $^{234}$U and $^{238}$U dpm/day. The ratios of $^{234}$U/$^{238}$U were close to 1. The absorption calculated for 6 individuals was 0.65% for $^{234}$U and 0.87% for $^{238}$U; the difference is probably related to counting error for the isotopes in urine. The mean, 0.76%, is taken as the best estimate of GI absorption in these persons.

[d] Absorption is taken as (urinary U/day)/(apparent intake).

Table A-5. Estimated gastrointestinal absorption of U in man, based on environmental data.

| Estimated U intake (µg U/kg/day) | GI absorption (%) | Method and references |
|---|---|---|
| 0.025 | 7.8 | Urine analysis, U.S. dietary survey (We60, We67) |
| 0.15 | 1.6 | Analysis of urine and local diet and water (Ya68, Mas71a,b,c,d) |
| 0.34 | 0.76 | Analysis of urine and feces of 6 subjects (Fish63) |
| 0.76 | 2.2 | 2 analyses of urine and feces of one subject (So80) |

Table A-6. Distribution, excretion and retention parameters of U and Ca in Reference Man.

| | Ca[a] | U[b] |
|---|---|---|
| Body content | 1000 g | 46 μg |
| Bone | 995 g | 25 μg |
| Soft tissues | 5 g | 13 μg |
| Daily intake | 1.1 g | 1.75 μg |
| Intestinal absorption (%) | 34 | ? |
| Retention in body (% at 10 days after injection) | 73 | 20[c] |

[a] Ca metabolic and distribution data from He64a,b; ICRP73,74; Spi68.

[b] U metabolic data from Tables B-1 and B-2.

[c] U retention data from Ba48; Hu73; Ber57.

Table A-7. Linear regression analysis of dependence of U absorption on U intake.

| Animal[a] (No. of studies) | Scale<br>U absorption | <br>U intake | Regression equation[b]<br>U absorption (%) = | Correlation coefficient (r) |
|---|---|---|---|---|
| rat (13) | linear | linear | $0.085 - 5.2\text{x}10^{-8}$ I | -0.174 |
| | | linear | log | 0.16 - |
| 0.02 log I | -0.351 | | | |
| | | log | log | $0.11\ \text{I}^{-}$ |
| 0.06 | -0.353 | | | |
| all others | linear | linear | $2.2 - 1.3\text{x}10^{-4}$ I | -0.323 |
| (10) | | linear | log | 2.6 - |
| 0.6 log I | -0.581 | | | |
| | | log | log | $1.8\ \text{I}^{-}$ |
| 0.12 | -0.705 | | | |
| (9) | | log | log | $1.4\ \text{I}^{-}$ |
| 0.08 | -0.601 | | | |

[a] Data from Tables A-2 and A-5.

[b] I is expressed in µg/day intake/kg of body weight.

Table B-1: Uranium in skeleton of persons with no known occupational exposure

| Source of samples | Skeletal parts measured | Reported U concentration ng U/g ash[a] | U in Reference Man skeleton<br>Total U (µg)[b] | | Reference and method |
|---|---|---|---|---|---|
| **United States** | | | | | |
| U.S. (?) | femur | 0.004 µg $^{238}$U/g ash | 4.0 | 11.2 | Rl 67; |
| Illinois | variety of bones | 0.022 µg U/g ash | 22. | 61.6 | We67; fluorometry |
| Illinois | same samples | 8.6 ng U/g ash | 8.6 | 24. | We76; fluorometry and mass spectrometry |
| New York City | vertebral bodies | 0.32 ng U/g wet | 2.3 | 6.4 | We76:fluorometry |
| 3 U.S. cities | vertebral ash | 0.6 dpm $^{238}$U/kg ash | 0.81 | 2.3 | Fi80; $^{238}$U alpha spectr. |
| Wisconsin | variety of bones | 0.02 µg U/g ash | 20. | 56. | Li81; $^{238}$U alpha spectr. |
| Colorado and Pennsylvania | vertebral bodies | 0.5 pCi $^{238}$U/kg wet[c] | 10.6 | 38.8 | Sin83; $^{238}$U alpha spectr. |
| New York State | long bone cortex | 21 pg $^{235}$U/g wet[d] | 4.6 | 12.9 | Sc81; NIAR |
| 5 U.S. regions[e] | vertebral bodies[f] | 199 fCi $^{234}$U/kg wet<br>22 fCi $^{235}$U/kg wet<br>150 fCi $^{238}$U/kg wet | 3.8 | 10.6 | Fr80,83; $^{234,235,238}$U alpha spectr. |
| **Other Countries** | | | | | |
| United Kingdom | variety of bones | 2.03 x $10^{-8}$g U/g ash[g] | 20.3 | 56.8 | Ha71, 72; delayed n |
| Japan | ribs | 6 ng U/g ash | 6.0 | 16.8 | No70; delayed n |
| India | not specified | 4.9 ng U/g ash | 4.9 | 13.7 | Ga70; |
| Nepal | vertebral bodies[f] | 11.8 dpm $^{238}$U/kg ash | 16.0 | 44.8 | Fi83; $^{238}$U alpha spectr. |
| Australia | vertebral bodies and ribs | 3.65 dpm $^{238}$U/kg ash | 4.9 | 13.7 | " " |

[a] Conversion factors used to obtain U concentration as ng U/g ash: Uranium composition and activity— 1 pCi = 2.22 dpm; specific activity of $^{238}U$ = 7.42 x $10^5$ dpm/g, only data for $^{238}U$ were used; abundance of $^{235}U$ = 0.007196. Factors related to bone composition— Ash fractions of cortical and cancellous human bone are 0.639 and 0.599, respectively (Go64a,b). All samples of vertebrae are assumed to have been obtained at routine autopsies, and therefore are vertebral body only. The ash fraction - 0.108 g ash/g wet vertebral body was used for the Colorado-Pennsylvania set (the composition of general hospital autopsy populations, obtained from the data tables of McI79 is as follows: 20 to 40 years 10.5%, 40 to 60 years 35%, more than 60 years 54.5%). The ash fraction = 0.14 g ash/g wet vertebral body was used for the New York City samples, which in the McI79 series, appear to be a coroner's population with a composition as follows: 20 to 40 years 50%, 40 to 60 years 45%, more than 60 years 5%. The age-related ash fractions of vertebral body were calculated from data given by Arnold and Wei (Ar72). The calculated value of 0.14 for the ash fraction of vertebral wedges of young adults is supported by the measured ash fraction (0.143 g ash/g wet vertebral wedge) for 4 large composites of vertebral wedge obtained after acute deaths of persons 20 to 25 years old in 4 separate geographic regions of the U.S. (Br80).

[b] The Reference Man skeleton contains 2800 g ash.

[c] One set of very high U concentrations (in soft tissues as well as bone) was suggestive of an unusual U exposure, and has been omitted.

[d] Only results from long bone cortex were used. The very much higher values obtained for cancellous bone may have been from contamination with $^{239}Pu$, the youth of the subject, and/or the presence of Cushing's disease.

[e] Five U.S. Environmental Protection Agency administrative regions: I, 6 New England states; II, New York, New Jersey, and Puerto Rico; III, 5 Central Atlantic states; V, 6 Great Lakes states; IX, California, Nevada, Arizona, and Hawaii.

[f] The weighted mean for all samples of bone.

[g] Only results of samples from persons more than 13 years old have been included.

Table B-2: U in soft tissues of persons with no known occupational exposure

| Tissue | Reported U Concentrations (ng U/g wet tissue) | Best value[a] |
|---|---|---|
| Liver | 0.25 (Ha72), 0.13 (We76), 0.30 (Sin83)[b] | 0.23 |
| Lung | 1.0 (We67), 0.53 (We76), 1.7 (Sin83)[b] | 1.08 |
| Kidney | 0.24 (We76), 0.63 (Sin83)[b] | 0.44 |
| Whole blood | 0.72 (Ham70b)[c], 0.57 (We76), 0.11 (Lu70) | 0.46 |
| Red cells | 0.15 (Lu70) | |
| Plasma | <0.08 (Lu70) | |
| Muscle | 0.19 (Ha72) | 0.19 |
| Heart | 0.16 (Ha72) | 0.16 |
| Spleen | 0.42 (Sin83)[b] | 0.42 |
| Gonad | 0.60 (Sin83)[b] | 0.60 |
| TB Lymph Nodes | 24.8 (Sin83)[b] | 24.8 |
| Fat | 0.60 (Ha72) | 0.60 |

---

[a] Best value is the arithmetic mean of the mean values reported by the different analysts; the value for blood includes separated red cells and plasma (Lu70).

[b] The values reported as dpm $^{238}U$/kg wet tissue (Sin83) have been recalculated as in the footnotes to Table B-1.

[c] Only the data for U.K. samples.

Table B-3: Estimation of U content of soft tissues of Reference Man.

| Tissue | U Concentration (ng/g wet weight) | Reference Man Tissue Weight (g) | U Content (μg) |
|---|---|---|---|
| Liver | 0.23 | 1,800 | 0.41 |
| Lung | 1.08[a] | 1,000 | (1.08)[b] |
| Kidney | 0.44 | 310 | 0.14 |
| Whole blood | 0.46 | 2,600[c] | 1.2 |
| Muscle | 0.19 | 28,000 | 5.3 |
| Heart | 0.16 | 330 | 0.053 |
| Spleen | 0.42 | 180 | 0.08 |
| Gonad | 0.60 | 35[d] | 0.021 |
| TBLN | 24.8[e] | 15 | (0.37) |
| Fat | 0.6[f] | (13,500) | (8.1) |
| Res. Soft Tissue[f] | | 32,000 | 5.6 |
| Total (adjusted)[b] | | | 13 |

[a] About 85% of lung U is considered the residue of inhalation.

[b] Adjusted total soft tissue does not include values enclosed in parentheses.

[c] Assuming only one-half of blood volume is drained from tissues at autopsy.

[d] Two testes.

[e] U in TBLN is considered to have been inhaled.

[f] See text.

Table C-1. Parameters of two-compartment models of U metabolism in human kidney.

| | $f_1$ | $f_{21}$ | $T_1$ (d) | $f_{22}$ | $T_2$ (d) | $A_w/m$ |
|---|---|---|---|---|---|---|
| ICRP(1979) | 0.05 | 0.12 | 6 | 0.00052 | 1500 | $3.5\text{x}10^{-4}$ |
| ICRP(1979) modified $f_1$ | 0.014 | 0.12 | 6 | 0.00052 | 1500 | $9.8\text{x}10^{-5}$ |
| Sp73 | 0.01 | 0.11 | 15 | 0 | 0 | $7.7\text{x}10^{-5}$ |
| Sp73 (modified $f_1$) | 0.1 | 0.11 | 15 | 0 | 0 | $7.7\text{x}10^{-4}$ |
| | 0.05 | 0.11 | 15 | 0 | 0 | $3.8\text{x}10^{-4}$ |
| | 0.03 | 0.11 | 15 | 0 | 0 | $2.3\text{x}10^{-4}$ |
| Committee Consensus | 0.014 | 0.11 | 15 | 0 | 0 | $1.1\text{x}10^{-4}$ |

Table C-2. Parameters of two-compartment models of U metabolism in human skeleton.

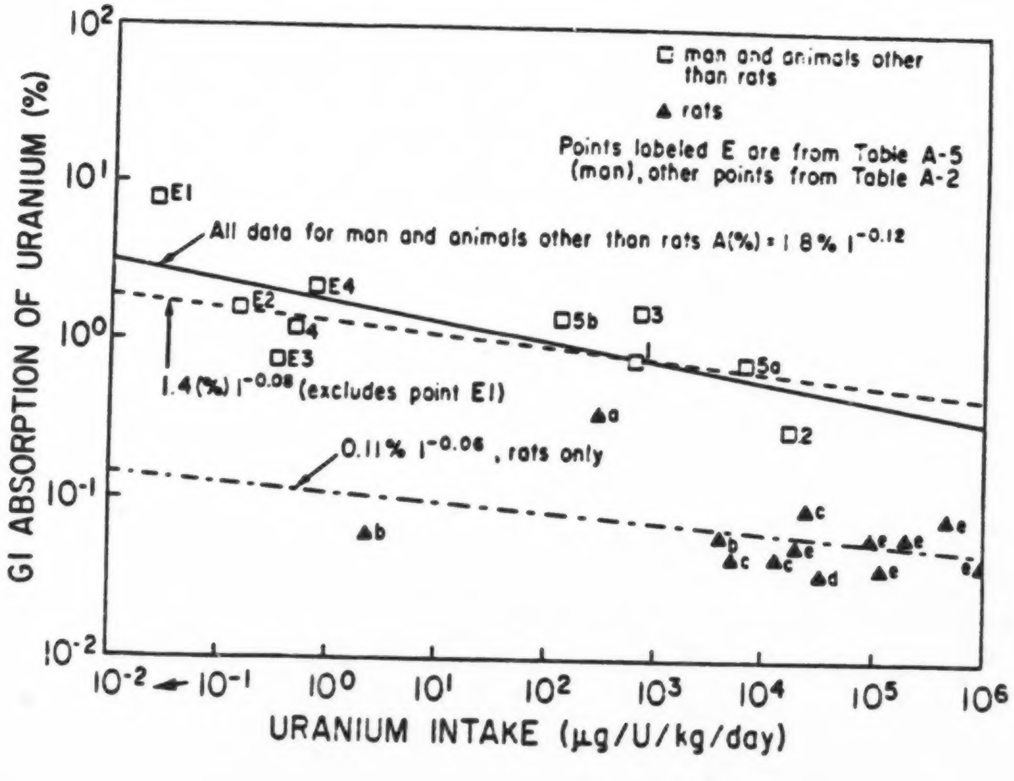
| | $f_1$ | $f_{21}$ | $T_1$(d) | $f_{22}$ | $T_2$(d) | $A_\infty/I$(d) (days) |
|---|---|---|---|---|---|---|
| ICRP (1979) | 0.05 | 0.2 | 20 | 0.023 | 5000 | 8.3 |
| modified $T_1$ | 0.05 | 0.2 | 300 | 0.023 | 5000 | 12.5 |
| modified $T_1$, $f_{21}$, $f_{22}$ | 0.05 | 0.1 | 300 | 0.1 | 5000 | 26.5 |
| Cothern et al. (Co83b) | 0.2 | 0.2 | 20 | 0.023 | 5000 | 33 |
| modified $f_1$ | 0.01 | 0.2 | 300 | 0.023 | 5000 | 1.7 |
| Committee Consensus* | 0.014 | 0.1 | 300 | 0.1 | 5000 | 10.7 |

---

* This model predicts 17 times greater amounts of U in cortical (compartment 2) vs. trabecular (compartment 1) bone, which seems at variance with reported data (Ha72, We67) on relative concentration of U in cortical and trabecular bone. No single model or combination of parameters chosen is completely consistent with the experimental data.

Caption

Figure A-1. Gastrointestinal absorption of U (%) plotted as a function of U intake (μg U/kg/day). Numbers within the symbols are the same as the numerical order of appearance of the experimental results in Table A-2. The circled alphabetic notations are gastrointestinal absorption from environmental data of We67 and So80. Results from experiments in rats and other experiments in which U ingested was more than $10^3$ μg U/kg/day have been omitted from the fits to curves 1 and 2 as unlikely to be encountered in environmental exposures.



□ man and animals other than rats
▲ rats
Points labeled E are from Table A-5 (man), other points from Table A-2
All data for man and animals other than rats A(%) = 1.8% $I^{-0.12}$
1.4(%) $I^{-0.08}$ (excludes point E1)
0.11% $I^{-0.06}$, rats only
E1
E2
E3
E4
4
5b
3
5a
2
a
b
c
d
e
GI ABSORPTION OF URANIUM (%)
$10^{2}$
$10^{1}$
$10^{0}$
$10^{-1}$
$10^{-2}$
$10^{-2}$ ← $10^{-1}$
$10^{0}$
$10^{1}$
$10^{2}$
$10^{3}$
$10^{4}$
$10^{5}$
$10^{6}$
URANIUM INTAKE (μg/U/kg/day)

## COMMITTEE ON THE HEALTH EFFECTS AND RISK DUE TO THE INTAKE OF NATURALLY OCCURING ALPHA PARTICLE EMITTERS

Chairman: Charles W. Mays
Recorder: Frederick Hodge
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Andrew F. Stehney

# CANCER RISK FROM THE LIFETIME INTAKE OF RADIUM AND URANIUM ISOTOPES*

Charles W. Mays
Radiobiology Division, Bldg. 351, U. of Utah
Salt Lake City, Utah 84112

Robert E. Rowland
Past Associate Director, Argonne National Lab
Present address; Box 281B, RR #1, Princeton, KY 42445

Andrew F. Stehney
Center for Human Radiobiology, Argonne National Lab
Argonne, Illinois 60439

ABSTRACT: From extensive human data on the induction of skeletal cancers (bone sarcomas and carcinomas of the head sinuses) by $^{226}$Ra, $^{228}$Ra, and $^{224}$Ra, the cumulative lifetime risk to 1,000,000 people, each ingesting 5 pCi of a radium isotope per day, was calculated to be 9 bone sarcomas plus 12 head carcinomas for $^{226}$Ra, 22 bone sarcomas for $^{228}$Ra, and 1.6 bone sarcomas for $^{224}$Ra. Assuming that the risk per rad of average skeletal dose is equal for $^{226}$Ra and the uranium isotopes with half-lives exceeding 1000 years, and that the equilibrium skeletal content is 25 times the daily ingestion of $^{226}$Ra, but 11 times the daily ingestion of long-lived uranium, the cumulative lifespan risk to 1,000,000 persons, each ingesting 5 pCi per day of $^{233}$U, $^{234}$U, $^{235}$U, $^{236}$U or $^{238}$U, is estimated to be about 1.5 bone sarcomas. The uranium risk is not well established and additional research is needed on the metabolism of uranium in humans and its carcinogenicity in laboratory animals. These estimates assume linear dose-responses. However, if incidence varies with the square of dose, virtually no induced cancers would be expected from these levels of radioactivity.

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## INTRODUCTION

We made this analysis at the request of C. Richard Cothern and William L. Lappenbusch of the Office of Drinking Water, U.S. Environmental Protection Agency, to provide the best presently available scientific information on the cancer risk from the daily ingestion of radium and uranium. The predicted cancer risk can then be compared to the financial costs and possible adverse health effects of procedures to decrease the radium or uranium concentrations in a local water supply.

In this paper we will estimate the lifetime risk of skeletal cancers in humans from the daily intake of $^{224}$Ra, $^{226}$Ra, and $^{228}$Ra. Using the risk coefficient for $^{226}$Ra-induced bone sarcomas, the lifetime risk will then be calculated for the daily intake of $^{233}$U, $^{234}$U, $^{235}$U, $^{236}$U, and $^{238}$U. The chemical toxicity of uranium is presented in a companion paper (Wr 84).

## TYPES OF CANCER INDUCED BY RADIUM ISOTOPES IN MAN

At low and medium doses of internally-deposited radium, the most severe biological damage is cancer arising from skeletal tissue. For $^{226}$Ra, the following two types of malignancy are induced: (1) Bone sarcomas (mostly osteosarcomas, fibrosarcomas, and chondrosarcomas), and (2) head carcinomas (carcinomas of the paranasal sinuses and the mastoid air cells).

Among some 3700 located U.S. persons* who were exposed to $^{226}$Ra and $^{228}$Ra intake by dial painting, medical administration, and other means, a

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*Different subgroupings of this population have been analyzed by the following quoted authors: Spiers, *et al.* (83), Rowland, *et al.* (83), and Stebbings, *et al.* (84). We give the composition of each subgroup as it is discussed in the text.

total of 85 cases of bone sarcoma and 36 cases of head carcinoma had been observed as of 31 December 1982 (Gu 83). However, no head carcinomas have yet been observed in the follow-up of 2324 German patients injected with $^{224}$Ra, although 55 have developed bone sarcomas (May 84, Wi 83). Similarly, no head carcinomas have been observed in persons internally contaminated with $^{228}$Ra, unless the dose from $^{226}$Ra was also high (Ev 66, Ro 78). This suggests that when $^{226}$Ra decays to $^{222}$Rn within the body, the accumulation of $^{222}$Rn gas in the head cavities is the major inducer of these carcinomas (Ev 66, Ro 78, Ma 84). For pure $^{224}$Ra and $^{228}$Ra, which do not produce $^{222}$Rn gas, the risk from head carcinomas is regarded as trivial compared to the risk from bone sarcomas.

For α-emitters deposited in mineral bone, the risk from radiation-induced leukemia in humans has been insignificant relative to that from bone sarcoma. In the subgroup of 2940 located males and females who were radium dial workers before 1970, there were 63 cases of bone sarcoma observed compared to 1 case expected naturally, in contrast to 10 cases of leukemia observed compared to 9 cases expected naturally (Spier 83, Ro 83). Furthermore, 4 of the 10 observed leukemias were chronic lymphocytic leukemia, a type of leukemia not found to be increased in any study of irradiated people (NAS 80).

The lifespan risk coefficient for leukemia-induction by α-particles in humans is very much lower than obtained by multiplying the ICRP risk coefficient for sparsely-ionizing radiation by a quality factor of 20 (ICRP 77).

$$\begin{matrix}\text{ICRP risk coefficient}\\ \text{for } \alpha\text{-particles}\end{matrix} = \left[\frac{20 \text{ leukemias}}{10^6 \text{ person}\cdot\text{rem}}\right]\left[\frac{20 \text{ rem}}{\text{rad}}\right] = \frac{400 \text{ leukemias}}{10^6 \text{ person}\cdot\text{rad}} \quad (1)$$

Spiers, *et al.* (Spier 83), have calculated the α-particle dose to red marrow in a special subgroup of 693 located women who were dial workers before 1930, measured while living, surviving without bone sarcoma for 2 years after their first measurement, and alive in 1957. Their α-particle dose to red marrow averaged about 25 rads. Assuming a similar average dose to the total measured and unmeasured subgroup of 1285 located women who were dial workers prior to 1930, the ICRP risk coefficient would predict about 13 cases of radiation-induced leukemia among these 1285 women.

$$[1285 \text{ persons}][25 \text{ rad}]\left[\frac{400 \text{ leukemias}}{10^6 \text{ person}\cdot\text{rad}}\right] = 13 \text{ leukemias} \quad (2)$$

The total number of predicted leukemias would then have been about 18 cases (13 radiation-induced plus 5 naturally-expected)(Spier 83). However, the prediction of 18 leukemias is strongly rejected by the observation of only 4 leukemias among these 1285 women. But if the risk coefficient were 10-fold lower, the predicted total of about 6 leukemias (1 radiation-induced plus 5 naturally-expected) would be in reasonable agreement with the observed 4 leukemias.

Independent evidence that the ICRP risk coefficient for α-particle-induced leukemia is about 10 times too high comes from Robin Mole's analysis of leukemias in the European Thorotrast patients (Mo 78). There were 44 leukemias observed among 4000 Thorotrast patients, injected with an average of 25 ml of Thorotrast, for which the dose rate to red marrow was 9 rads per year, giving a dose of 270 rads in 30 years of follow-up.

$$\begin{array}{l}\text{Leukemia risk coefficient}\\ \text{for Thorotrast } \alpha\text{-particles}\end{array} = \frac{44 \text{ leukemias}}{(4000 \text{ persons})(270 \text{ rad})} = \frac{40 \text{ leukemias}}{10^6 \text{ person}\cdot\text{rad}} \quad (3)$$

Why is the observed risk coefficient for leukemia induction by α-particles in humans so much lower than the ICRP risk coefficient given in Equation 1? Possible explanations might include: (1) Nonuniform irradiation which allows nonirradiated (undamaged) stem cells to replace cells killed by irradiation, (2) low frequency of potentially leukemogenic cells in the regions most heavily irradiated by α-particles such as regions near the bone surfaces or near the phagocytic cells that take up colloidal Thorotrast, (3) a leukemia RBE less than 20 for uniform α-particle vs. sparsely ionizing radiation at low dose rate, (4) a lifespan risk coefficient less than 20 leukemias/$10^6$ person•rem for sparsely ionizing radiation at low dose rates, or (5) other factors. Regardless of which theoretical possibility is correct, it is now firmly established by direct observation of humans exposed to graded levels of α-radiation emitted from mineral bone that the risk from induced leukemia is very small compared to the risk from induced bone sarcomas (Spier 83, May 83). We have given considerable attention to the risk from leukemia, primarily because a recent calculation suggested that the lifetime ingestion of $^{226}$Ra might induce more leukemias than bone cancers (Co 83). However, the opposite is indicated by actual observation on the effects of radium isotopes in humans (Spier 83, Ro 83, May 83).

Multiple myeloma has occurred in 6 of the 1285 located women who were dial workers prior to 1930, compared to 2.1 cases naturally-expected (Steb 84). While the 3-fold increase is statistically significant ($P = 0.042$), the increase is not necessarily due to internally-deposited $^{226}$Ra and $^{228}$Ra. The external γ-ray exposure from the radium paint pots has been estimated at 8-20 rad/year (Steb 84). Of the 6 myeloma cases, 4 worked as dial painters at least 50 weeks, one worked only 2 weeks, and the duration of employment for the remaining one is unknown (Steb 84). Even if the 4 excess cases of myeloma were caused by skeletally-deposited radium, induced myeloma would be only 6% as frequent as the 63 cases of bone sarcoma observed among these 1285 women. Among the 2324 German $^{224}$Ra patients, the 1 case of multiple myeloma (plasmacytoma) was only 2% as frequent as the 55 cases of bone sarcoma observed among these patients (Ma 84, Wi 83). Multiple myeloma appears to be only slightly inducible by radium in human bone.

For the 1285 located women who were dial workers before 1930, Stebbings, *et al.* (Steb 84), have compared the numbers of observed vs. expected cancers for the 10 following types of highest naturally-expected frequency: breast, colon, lung, stomach, pancreas, cervix uteri, corpus uteri, rectum, leukemia, and liver. For none of these cancers was the observed incidence significantly different ($P<0.05$) from the natural expectation based on rates in the relevant counties. Summing the 10 sites, there were 101 observed cancer cases vs. 94 expected, based on country rates. The excess of only 7 cases indicates that the internally-deposited radium and the external $\gamma$-ray exposure received by these women were relatively ineffective in inducing nonskeletal cancers.

It is clear that the observed 63 bone sarcomas and 23 head carcinomas represent the vast majority of radium-induced cancers among the 1285 located women who were radium dial workers prior to 1930, although a small number of additional cancers might possibly have been induced in other tissues (Fin 69, Steb 84).

Similarly, among 612 adult German patients of known dose, injection span, and health status (as of June 1974), 38 soft-tissue neoplasma were observed compared to about 33-40 naturally-expected cases during the follow-up averaging 17 years for the adults (Spies 78). Among a similar 204 patients injected as juveniles and followed an average of 20 years, 5 soft-tissue neoplasms were observed, similar to natural expectation for their ages (Spies 78). If an excess of soft-tissue neoplasms had occurred, it was small relative to the 47 of these patients of known dose that developed bone sarcoma. (Among the 899 patients of known and unknown dose in the Spiess series, 53 have developed bone sarcomas.)

## DOSE-RESPONSE RELATIONSHIPS

### 1. Head carcinomas

The incidence rate $\underline{I}$ for head carcinomas, due to $^{226}$Ra intake to blood, was fitted to a variety of dose-response relationships of the general form $I = (C + \alpha D + \beta D^2)e^{-\gamma D}$ for the measured female dial workers employed before 1930 (Ro 78). A minimal latent period of 10 years was assumed. Based on Chi-squared statistics, the best fit (probability $P = 0.86$) was to the following linear relationship (Ro 78):

$$\begin{matrix}\text{Yearly incidence} \\ \text{(carcinomas/person·yr)}\end{matrix} = 1.6 \times 10^{-5}\ D \qquad (4)$$

where the radium intake to blood is $\underline{D}$ μCi $^{226}$Ra. The intake of $^{226}$Ra to blood was back-calculated from the measured body content of $^{226}$Ra at "t" days after intake using the Norris retention function (No 55), retention $R = 0.54\ t^{-0.52}$. The intake of $^{228}$Ra was disregarded, since head carcinomas have not been associated with $^{228}$Ra.

### 2. Bone sarcomas

Two groupings were made for analysis of bone sarcoma incidence in the measured female dial workers employed before 1950 (Ro 83).

Group A consisted of all measured female dial workers. Their years at risk were assumed to begin 5 years after the start of employment (allowing for a 5-year minimal latent period for bone sarcoma) and to continue until death, the diagnosis of a bone sarcoma, or the end of follow-up. Group A contained 42 bone sarcomas among 1468 persons averaging 40.0 years at risk. It may underrepresent persons who died early and were not measured for radium, but overrepresent those who ultimately suffered from the effects of radium and were identified and measured as a consequence.

Subgroup B, a part of Group A, was selected to eliminate persons possibly measured for radium as a consequence of their cancers. It included only the female dial workers who were measured for radium while living and excluded any person who died or was diagnosed with a bone sarcoma within 2 years after the first measurement, since the symptoms of bone sarcoma usually first appear within 2 years before diagnosis or death. Thus, the years of monitored risk for Subgroup B started 2 years after the first measurement and continued until death, the diagnosis of a bone sarcoma, or the end of follow-up. Subgroup B contained 13 bone sarcomas among 1257 persons averaging 9.4 years at monitored risk.

For Group A, the incidence rate for bone sarcomas was best fit by Chi-squared analysis (P = 0.73) by the following dose-squared - exponential equation (Ro 83):

$$\begin{array}{c}\text{Yearly incidence}\\ \text{(sarcomas/person}\cdot\text{yr)}\end{array} = 7.0 \times 10^{-8}\, D^2\, e^{-0.0011\, D} \qquad (5)$$

Where for bone sarcomas, D is the intake to blood in μCi $^{226}$Ra plus 2.5 times the intake in μCi $^{228}$Ra, since 1 μCi $^{228}$Ra with high retention of daughters was estimated to be equivalent for bone sarcoma-induction to about 2.5 µCi $^{226}$Ra with low retention of daughters (Ro 78).

No bone sarcomas have yet been identified among any of the exposed measured persons with radium intakes to blood below 50 µCi (Ro 83). Among the measured 1321 female dial workers below 50 µCi intake, the preceding equation plus the natural incidence predict 0.67 total expected bone sarcomas (0.29 induced plus 0.38 natural). For an expectation of 0.67 sarcomas, zero sarcomas could occur with a 51% chance ($P = 0.51$).

At low values of intake, the exponential in Equation 5 approaches unity, and the calculated yearly incidence from $^{226}$Ra varies as the square of the intake. For an intake of 1 µCi $^{226}$Ra, the calculated yearly incidence of $7 \times 10^{-8}$ is less than 1% of the natural yearly incidence of about $1 \times 10^{-5}$. At lower doses the calculated yearly incidence from $^{226}$Ra rapidly approaches zero. We will use zero risk as a lower limit for radium-induced bone sarcomas at low intakes.

For the epidemiologically more suitable Subgroup B, acceptable fits were observed for the following equations (Ro 83):

Linear Model ($P = 0.26$)

$$\frac{\text{Yearly incidence}}{\text{(sarcomas/person}\cdot\text{yr)}} = 2.0 \times 10^{-5}\ D \qquad (6)$$

Dose-Squared - Exponential Model ($P = 0.27$)

$$\frac{\text{Yearly incidence}}{\text{(sarcomas/person}\cdot\text{yr)}} = 18 \times 10^{-8}\ D^2\ e^{-0.0015\ D} \qquad (7)$$

Where for bone sarcomas, D is the intake to blood in µCi $^{226}$Ra plus 2.5 times the intake in µCi $^{228}$Ra.

The linear equation (Eq. 6) from Subgroup B predicts a total of 1.25 bone sarcomas (1.06 induced plus 0.19 natural) in the Subgroup B female dial workers with intakes to blood below 50 μCi. However, applying this equation to the 1321 female dial workers below 50 μCi in Group A, with their much longer assigned years at risk, predicts a total of 4.45 bone sarcomas (4.07 induced plus 0.38 natural). For an expectation of 4.45 sarcomas there is only a 1.2% chance that zero sarcomas would occur ($P = 0.012$). Hence, we regard linear equation (Eq. 6) $2 \times 10^{-5}$ D as a reasonable upper limit to the risk from radiation-induced bone sarcomas at low intakes.

We regard the actual risk from bone sarcoma at low intakes of radium to be somewhere between our upper limit (linear Eq. 6) and the lower limit of zero. Tentatively, we arbitrarily assume it to be midway between our upper and lower limits.

Provisional best estimate of bone sarcoma risk from $^{226}$Ra

$$\begin{array}{c}\text{Yearly incidence}\\ \text{(bone sarcomas/person·yr)}\end{array} = 1 \times 10^{-5}\ D \qquad (8)$$

The above provisional best estimate predicts 2.41 total expected bone sarcomas (2.03 induced + 0.38 natural) among the 1321 female dial painters of Group A with intakes below 50 μCi. For an expectation of 2.41 sarcomas there is a 9% chance that zero sarcomas would occur. Hence, Eq. 8 cannot be statistically rejected ($P > 0.05$).

It is possible that 1-or-more new bone sarcomas may occur in the low-intake persons under present surveillance. In fact, 1-or-more sarcomas at low-intake may already exist among the 24 known cases of bone sarcoma who were buried without an evaluation of radium content (Gu 83; see p. 162).

Therefore, it is important to exhume and measure as many as possible of the 24 buried but unmeasured bone sarcoma patients, and to continue to follow the living exposed persons. If no additional sarcomas are found, it would suggest that our provisional best estimate of bone sarcoma risk may be too high, whereas finding more sarcomas may suggest that our tentative best estimate is about right or perhaps too low. The present study is unique in that it offers the only known opportunity to resolve this question of scientific and humane importance.

## CUMULATIVE RISK FROM LIFELONG INTAKE OF RADIUM

The following cumulative risk estimates for long-lived $^{226}$Ra and $^{228}$Ra correspond to a constant concentration of radium in bone throughout life --- at the equilibrium concentration resulting from the adult intake to blood of 1 pCi of a radium isotope per day. The daily intake required to maintain a constant concentration of radium in bone may differ for infants, children, adolescents, and adults. The general finding that the $^{226}$Ra/Ca ratio in bone remains roughly constant with age (Fis 79, Ra 64) probably reflects a more-or-less constant ratio of $^{226}$Ra/Ca in the diet at environmental levels during much of the lifespan (Wr 77, Wr 84). Even the recent results of Muth and Glöbel (Mu 83), which suggest possible peaks in $^{226}$Ra concentration at about 1 and 10 years of age, correspond to an average lifespan concentration only about 20% higher than the average of their adult values. For simplicity, we assume that the daily intake of dietary $^{226}$Ra and $^{228}$Ra at environmental levels result in constant Ra/Ca ratios in bone. For long-lived $^{226}$Ra ($T_{\frac{1}{2}}$ = 1600 yr) and $^{228}$Ra ($T_{\frac{1}{2}}$ = 5.77 yr), one might expect the high rate of Ra accretion in the growing bones of a child to be opposed by the enhanced rate of Ra removal from resorbing bone.

However, short-lived $^{224}Ra$ ($T\frac{1}{2}$ = 3.62 days) should decay before appreciable resorption occurs. Therefore, we assume that skeletal concentrations of $^{224}Ra$ in children aged 0-15 and in adolescents, aged 16-20, respectively, would be 3 and 2 times the adult concentrations, based on the fractions of injected $^{224}Ra$ estimated to decay in bone (Spies 70).

Now the risks from the lifelong intakes to blood of 1 pCi Ra/day will be estimated for $^{226}Ra$ (head carcinomas and bone sarcomas), $^{228}Ra$ (bone sarcomas only), and $^{224}Ra$ (bone sarcomas only).

### $^{226}Ra$: Cumulative risk from HEAD CARCINOMA:

The average life expectancy in the U.S. has increased from 50 years in 1910, to 60 years in 1930, to 70 years in 1960, and was 74 years in 1980 (Ub 84). We assume it to be about 75 years in the mid-1980's. For a lifetime of 75 years, only the $^{226}Ra$ intake during the first 65 years is considered effective for the induction of observable head carcinomas because of the assumed minimal latent period of 10 years for these carcinomas (Ro 78).

$$\begin{array}{l}^{226}\text{Ra cumulative}\\ \text{intake to blood}\end{array} = \left(\frac{10^{-6}\ \mu\text{Ci}}{\text{day}}\right)\left(\frac{365\ \text{days}}{\text{yr}}\right)(65\ \text{yr}) = 0.0237\ \mu\text{Ci} \qquad (9)$$

The risk rate for observable head carcinomas builds up linearly from zero at age 10 years (the minimal latent period) to a maximum 65 years later at the end of life. From equations 4 and 9:

$$\text{Risk rate at end of life} = \left(\frac{1.6 \times 10^{-5}}{\mu\text{Ci}\cdot\text{yr}}\right)(0.0237\ \mu\text{Ci}) = \frac{0.380 \times 10^{-6}}{\text{yr}} \qquad (10)$$

$$\text{Av. risk rate during expression period} = \frac{0 + 0.368 \times 10^{-6}/\text{yr}}{2} = \frac{0.190 \times 10^{-6}}{\text{yr}} \qquad (11)$$

$$\text{Cumulative risk } (^{226}\text{Ra head carc.}) = \left(\frac{0.190 \times 10^{-6}}{\text{yr}}\right)(65\ \text{yr}) = 12 \times 10^{-6} \qquad (12)$$

In words, if 1 million persons each had the equivalent intake throughout life of 1 pCi $^{226}$Ra per day to blood, about 12 cases of induced head carcinoma would be predicted. For a natural incidence rate averaging $0.5 \times 10^{-5}$ per year, about 375 naturally-occurring head carcinomas should occur among 1,000,000 persons during their lifespan, averaging about 75 years.

### $^{226}$Ra: Cumulative risk from BONE SARCOMA:

For a lifetime of 75 years, only the $^{226}$Ra intake during the first 70 years is considered effective for the induction of observable bone sarcomas because of the assumed minimal latent period of 5 years for bone sarcomas (Ro 78).

$$^{226}\text{Ra cumulative intake to blood} = \left(\frac{10^{-6}\ \mu\text{Ci}}{\text{day}}\right)\left(\frac{365\ \text{days}}{\text{yr}}\right)(70\ \text{yr}) = 0.0256\ \mu\text{Ci} \qquad (13)$$

The risk rate from induced bone sarcomas builds up linearly from zero at age 5 years (the minimal latent period) to a maximum 70 years later at the end of life. From equations 8 and 13:

$$\text{Risk rate at end of life} = \left(\frac{1 \times 10^{-5}}{\mu\text{Ci}\cdot\text{yr}}\right)(0.0256\ \mu\text{Ci}) = \frac{0.256 \times 10^{-6}}{\text{yr}} \quad (14)$$

$$\text{Av. risk rate during expression period} = \frac{0 + 0.256 \times 10^{-6}/\text{yr}}{2} = \frac{0.128 \times 10^{-6}}{\text{yr}} \quad (15)$$

$$\text{Cumulative risk } (^{226}\text{Ra bone sar.}) = \left(\frac{0.128 \times 10^{-6}}{\text{yr}}\right)(70\ \text{yr}) = 9 \times 10^{-6} \quad (16)$$

In words, among 1 million persons so exposed, about 9 cases of induced bone sarcoma would be predicted. For a natural incidence rate, averaging $1 \times 10^{-5}$ per year, about 750 naturally-occurring bone sarcomas should occur among 1,000,000 persons during their lifespans, averaging 75 years.

A total of 21 $^{226}$Ra-induced cancers (12 carcinomas + 9 sarcomas) are predicted for this exposed population.

### $^{228}$Ra: Cumulative risk from BONE SARCOMA:

Following the above procedures, but assuming that each µCi of $^{228}$Ra is equivalent to 2.5 µCi $^{226}$Ra for bone sarcoma-induction (Ro 78), about 2.5 x 9 = 22 bone sarcomas should accumulate among the lifespan of 1,000,000 persons, each with an intake to blood of 1 pCi $^{228}$Ra per day.

### $^{224}$Ra: Cumulative risk from BONE SARCOMA:

The intravenous injection of 1 µCi $^{224}$Ra gives a cumulative α-particle dose of about 0.2 rad averaged over the 7-kg marrow-free skeleton of adult reference man (Spies 70). Thus, the adult intake to blood of 1 pCi $^{224}$Ra/day or 365 x $10^{-6}$ µCi/year, gives an average skeletal dose rate of 73 x $10^{-6}$ rad/year to adult man. We assume, however, that the skeletal concentrations of short-lived $^{224}$Ra are 3 times higher in children under age 16, and 2 times higher in adolescents 16 thru 20 years than in adults, based on the fractions of intravenously-injected $^{224}$Ra estimated to decay in juvenile, adolescent, and adult bone (Spies 70). The dose within each age interval is:

| Age | Years in interval | Dose rate (rads/yr) | Rads in interval |
|---|---|---|---|
| under 16 | 16 | $3 \times 73 \times 10^{-6}$ | 0.00350 |
| 16 thru 20 | 5 | $2 \times 73 \times 10^{-6}$ | 0.00073 |
| 21 to 70 | 49 | $1 \times 73 \times 10^{-6}$ | 0.00358 |
| Cumulative | 70 | | 0.00781 |

Using the cumulative risk coefficient of 200 bone sarcomas/$10^6$ person·rad of average skeletal dose from $^{224}$Ra, which life-table analysis has shown to be fairly constant for injection ages ranging from 1-70 years (May 83):

$$\begin{matrix}\text{Cumulative risk} \\ (^{224}\text{Ra bone sar.})\end{matrix} = \left(\frac{200 \text{ bone sar.}}{10^6 \text{ person}\cdot\text{rad}}\right)(0.00781 \text{ rad}) = 1.6 \times 10^{-6} \qquad (17)$$

Among 1 million persons so exposed, there should be an expectation of about 1.6 induced bone sarcomas. Alternatively, only 1.0 induced sarcoma should be expected if the concentration of $^{224}$Ra in bone remained constant at the adult equilibrium concentration.

For 1 pCi/day input to blood for 1 million people, the predicted 1.6 bone sarcomas for $^{224}$Ra is much lower than the predicted 9 bone sarcomas for $^{226}$Ra. This is primarily because the short 3.62-day half-life of $^{224}$Ra limits the amount of radiation dose it can deliver. However, the short half-life of $^{224}$Ra causes much of it to decay on bone surfaces where it has enhanced effectiveness per rad of average skeletal dose.

The gut uptake of 20% for radium by adult persons was carefully established (Mal 69) by the absorption of short-lived $^{224}$Ra from mock radium dial paint ingested by elderly adult volunteers. Thus, the ingestion of 5 pCi Ra results in the intake to blood of 1 pCi Ra. If the concentration of radium in a local water supply is 5 pCi Ra per liter, and 1 liter per day of local water is consumed, the intake to blood would be 1 pCi Ra per day from this source.

## DAILY FLUID INTAKE FROM THE LOCAL WATER SYSTEM

We assume an average adult consumption of 1 liter per day from the local water system (tap water), based on the average reported consumption of 32.7 ounces (0.97 liters) per day from a survey of some 200 residents of New Mexico, published by Johnathan M. Mann, State Epidemiologist for the State of New Mexico (Man 83). Consumption of local water includes the direct drinking of tap water, the tap water in home-prepared drinks such as coffee and tea, and the tap water in locally-prepared foods such as soups and cooked vegetables. It excludes water from sources outside the local water supply such as milk, bottled drinks, canned food, and the water formed within the body by the metabolic oxidation of food.

The daily drinking of tap water alone by reference persons is given in ICRP Publication 23 as 150 ml for adult man, 100 ml for adult woman, and 200 ml for a 10-year-old child (ICRP 75). However, the total fluid intake from the local water supply exceeds the direct drinking of tap water.

The Environmental Protection Agency commonly assumes a daily consumption of 2 liters of water per day (Co 83). This was based on an upward rounding from the average per capita water (liquid) consumption of 1.63 liters per day, as calculated from a survey of 9 different literature sources (NAS 77). Examination of these sources revealed that the 1.63 liters per day included not only local tap water but nonlocal sources such as bottled drinks. Thus, from the local water supply alone, a lower average fluid intake of about 1 liter per day seems reasonable, although improved evaluations of the true average are needed.

Assuming a linear dose-response relationship for cancer induction at environmental levels, the population risk is determined by the average intake, not by the maximum intake to the highest-exposed individual. For example, for a local water supply containing 100 pCi $^{226}$Ra per liter, the predicted number of induced cancers is the same in a population of 1,000,000 persons, each drinking 1 liter per day from the system, as in 1,000,000 similar persons of whom 500,000 drink 0.5 liters per day and the other 500,000 drink 1.5 liters per day.

In contrast, to prevent a presumed threshold type of chemical toxicity, it is appropriate to consider a higher-than-average daily intake such as the 1.7 liters per day used by Wrenn, *et al.*, to evaluate the threshold for chemical damage to the kidney from uranium (Wr 84).

To limit the risk of radionuclide-induced cancer, some regulators may prefer inflating the average daily intake from a local water supply to 2 liters per day to provide a "hidden" safety factor. We prefer to estimate the risks as realistically as possible. We believe that when safety factors are desired, they should be given openly rather than be disguised.

## ESTIMATED RISK FROM LIFELONG INTAKE OF URANIUM

Whereas the risk from the radium isotopes, $^{226}$Ra, $^{228}$Ra, and $^{224}$Ra, is based on the observed toxicity in humans and can be expressed per μCi intake, no direct information exists on cancer-induction by uranium in people. Therefore, the risk from uranium will be inferred from the risk for long-lived, bone-volume-seeking $^{226}$Ra.

For the lifespan dietary intake of uranium at environmentals levels, most of the retained uranium in the skeleton is uniformly distributed throughout the volume of mineralized bone (Wr 84). Thus, the bone sarcoma risk per rad of average skeletal dose from bone-volume-seeking uranium isotopes with half-lives exceeding 1,000 years, is more likely to resemble that from long-lived, bone-volume-seeking $^{226}$Ra (which produces no bone-surface-seeking daughters) than from either $^{224}$Ra (much of which quickly decays while still on bone surfaces) or $^{228}$Ra (which decays to surface-seeking $^{228}$Th and $^{224}$Ra).

In adult and adolescent males, the skeletal content of $^{226}$Ra at equilibrium is about 25 times the daily ingestion (Steh 56, Wr 84). Thus, the adult ingestion of 5 pCi $^{226}$Ra per day should produce an equilibrium skeletal content of about 125 pCi $^{226}$Ra. The $^{226}$Ra, with 30% retention of its daughters, $^{222}$Rn, $^{218}$Po, $^{214}$Po, and $^{210}$Po (ICRP 79), released a summed 12.11 MeV of α-particle energy per $^{226}$Ra disintegration.

For the five uranium isotopes with half-lives exceeding 1000 years, the individual half-lives and α-energies per decay are as follows (ICRP 83):

| Isotope | Half-life (yr) | α-energy (MeV) |
|---|---|---|
| $^{233}$U | $1.59 \times 10^5$ | 4.82 |
| $^{234}$U | $2.44 \times 10^5$ | 4.76 |
| $^{235}$U | $7.04 \times 10^8$ | 4.40 |
| $^{236}$U | $2.34 \times 10^7$ | 4.50 |
| $^{238}$U | $4.47 \times 10^9$ | 4.19 |

The individual α-energies cluster closely around the group average of 4.5 MeV, which we will use.

For consistency with our example for radium, consider the ingestion of 5 pCi of uranium per day. The equilibrium skeletal content of long-lived uranium lies between 1 and 35 times the daily ingestion, with 11 as a best present estimate (Wr 84). Thus, in our example, the best present estimate of the equilibrium skeletal content would be 11 x 5 = 55 pCi of uranium.

The cumulative lifespan risk from the daily ingestion of 5 pCi uranium per day, can be computed from the cumulative lifespan risk of 9 bone sarcomas/$10^6$ persons each ingesting 5 pCi $^{226}$Ra per day (Eq. 13) and the equilibrium skeletal burdens and summed $\alpha$-energies:

$$\begin{array}{l}\text{Lifetime risk}\\ \text{(Long-lived U)}\end{array} = \left[\frac{\text{9 bone sar.}}{10^6\ \text{persons}}\right]\left[\frac{\text{55 pCi Uran. in skel.}}{\text{125 pCi }^{226}\text{Ra in skel.}}\right]\left[\frac{\text{4.5 MeV from Uran.}}{\text{12.11 MeV }^{226}\text{Ra + dau.}}\right]$$

$$= \frac{\text{1.5 bone sarcomas}}{10^6\ \text{persons}} \qquad (18)$$

In 1 million persons so exposed, about 1.5 cases of uranium-induced bone sarcoma are predicted, assuming that the ratio of skeletal content to daily ingestion is 11 (best present estimate). Since the true ratio is within the range of 1-35, the corresponding number of predicted bone sarcomas could range from 0.1-5. In contrast, about 750 naturally-occurring bone sarcomas are expected among these 1,000,000 persons during their lifetimes.

$^{232}$U has been omitted from these calculations because it decays to surface-seeking $^{228}$Th. The toxicity per rad from $^{232}$U with its decay products might be similar to that for $^{228}$Ra, which also decays to $^{228}$Th.

The build-up of $^{222}$Rn gas is not a problem with pure uranium isotopes, and thus the risk from head carcinomas is considered negligible compared to that from bone sarcomas. Head carcinomas are not a problem except when $^{226}$Ra deposits in bone.

Whereas the risk from induced soft-tissue cancers is known to be small for internally-deposited $^{226}$Ra, $^{228}$Ra, and $^{224}$Ra, the possible risk of soft-tissue cancers induced by uranium isotopes is unknown, but by analogy with radium is not predicted to be large.

The predicted numbers of induced cancers for radium and uranium isotopes, based on a linear dose-response, is shown in Table 1. However, if the true dose-response for α-particle-induced cancers in humans at low doses varies with the square of dose, virtually no cancers would be expected from environmental levels of radium or uranium.

## THE COST OF REMEDIAL MEASURES

There are two kinds of costs in remedial measures: (a) the direct financial cost, and (b) the biological cost which, in part, becomes a financial cost. Both are important and should be considered. Remedial action at environmental levels should not be taken unless the benefit of the remedial action exceeds the total cost of this action (financial and biological).

Specifically, an important practical consideration is to assess the total costs of alternative methods of removing radium from drinking water. One way is to use ion exchange columns that remove divalent cations such as $Mg^{++}$, $Ca^{++}$, $Sr^{++}$, $Ba^{++}$, and $Ra^{++}$ from water, replacing them with sodium ions, $Na^{+}$. But what is the health impact of increasing the sodium concentration in drinking water, especially on highly susceptible individuals such as persons with high blood pressure who must minimize their sodium intake? Is it proper to make a small reduction in an assumed cancer risk from radium at the expense of killing a much larger number from sodium-augmented cardiovascular disease? It would be much more difficult to detect a moderate numerical increase in deaths from cardiovascular disease, presently about 50% of U.S. deaths (Am 81) than from bone sarcomas and head carcinomas (only about 0.1% of U.S. deaths). We urge caution until the total costs are better known.

Recently the Environmental Protection Agency sponsored a symposium on Drinking Water and Cardiovascular Disease (Ca 80). It is our impression, that relative to the effects of radium at low doses, the health effects of sodium addition, while difficult to assess, could be substantially greater.

Often, it may be more feasible to switch to a less-contaminated source of drinking water than to remove the radioactivity from a local supply.

## RECOMMENDATIONS FOR FUTURE RESEARCH

1. Continue to follow the health of the U.S. radium persons internally contaminated with $^{226}$Ra and $^{228}$Ra, and the German patients injected with $^{224}$Ra, until virtually all of them have died. Until this is done, uncertainty will remain on the total health effects from radium isotopes in human beings.
2. Reliably establish the ratio between uranium in the skeleton at equilibrium and the daily ingestion of uranium.
3. Using laboratory animals, evaluate the bone sarcoma risk per rad from high-specific activity uranium such as $^{233}$U, and compare this risk with that from $^{226}$Ra. Is the assumption of equal risks per rad really valid?
4. Emphasize basic research on the nature of radiation-induction of cancer. An important endpoint should be better prediction of the actual effects at low doses, based on observed effects at higher doses.
5. Explore alternative ways to reduce intake of radioactivity by the population if, indeed, such reductions are to be seriously considered. The

biological, as well as engineering costs, of these alternatives should be established well enough that the decisions should have a net positive benefit.

## ACKNOWLEDGMENT

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TABLE 1. Best present estimates of induced cancers during the lifetimes of 1,000,000 persons, each with a constant skeletal concentration equal to the adult equilibrium level resulting from the daily ingestion of 5 pCi of a radium or uranium isotope.

| Radionuclide | $\left[\frac{\text{Bone sarcomas}}{10^6 \text{ persons}}\right]$ | $\left[\frac{\text{Head carcinomas}}{10^6 \text{ persons}}\right]$ |
|---|---|---|
| $^{226}$Ra | 9 | 12 |
| $^{228}$Ra | 22 | 0 |
| $^{224}$Ra | 1.6* | 0 |
| $^{233}$U | 1.5** | 0 |
| $^{234}$U | | |
| $^{235}$U | | |
| $^{236}$U | | |
| $^{238}$U | | |

*The skeletal concentration of short-lived $^{224}$Ra was assumed 3 times higher in children under 16 years of age, and 2 times higher in adolescents 16-20 years of age, than in adults.

**For these uranium isotopes, the estimated number of induced bone sarcomas/$10^6$ persons ranges from 0.1-5.0 with a best estimate of 1.5.

## COMMITTEE ON COMPLIANCE AND POLICY

Chairman: John E. Regnier

Recorder: Robert Sullivan

Committee Members: Richard Blanchard
Melvin W. Carter
Edward Cowan
David Duncan
James Martin

# COMPLIANCE AND POLICY ISSUES AND RECOMMENDATIONS RELATED TO REVISION OF THE NATIONAL INTERIM PRIMARY DRINKING WATER REGULATIONS FOR RADIONUCLIDES

John E. Regnier,
Alabama Rural Water Association
Montgomery, AL 36105,

Richard Blanchard
U.S. Environmental Protection
Agency
P.O. Box 3009
Montgomery, AL 36193,

Melvin W. Carter
Georgia Institute of Technology
Atlanta, GA 30332,

Edward Cowan
U.S. Environmental Protection
Agency
Seattle, WA 98101,

James Martin
2604 Bedford Road
Ann Arbor, MI 48104,

and

Robert Sullivan
U.S. Environmental Protection
Agency
Office of Radiation Programs
Washington, D.C. 20460

## ABSTRACT

This paper summarizes the deliberations and conclusions of the Compliance and Policy Committee of the National Workshop on Radiation in Drinking Water held in Easton, Maryland, May 24, 25 & 26, 1983. Prior to and during the workshop the Committee considered a total of 32 possible compliance and policy issues and determined that 22 were valid. The Committee developed positions on seven of these and these positions are presented herein. The remaining 25 issues which were considered are also listed with the Committee's evaluation of each indicated.

The purpose of this paper is to present the results of the deliberations of The Compliance and Policy Committee prior to and during the workshop. Prior to the workshop, this committee exchanged written and verbal communications on possible policy issues. From these exchanges and from input from other committees and EPA a composite list of tentative issues was drawn up and circulated for comment. During the workshop, this list was analyzed as follows:

1) Was a true issue involved rather than a matter requiring a technical assessment?
2) If a true issue was involved, was it of a policy matter or a technical matter and therefore, better addressed in a different committee?
3) Were there additional issues that needed consideration?

Subsequent to this analysis, the committee addressed those policy issues it felt were of highest priority and developed a policy statement and/or a recommendation on each.

In the remainder of this discussion, all issues considered are presented, an indication is given of whether the committee felt each was a valid policy issue, and policy statements are provided on those issues judged to be most pressing.

## POSSIBLE ISSUES

Prior to the workshop a tentative list of 29 possible issues had been identified. During the workshop, as a result of input from EPA, other committees, and observers, three additional issues were added. These issues, posed as questions, are tabulated in the Appendix.

As previously mentioned, the committee deliberated the merit of each question to decide if it required more than a simple technical assessment, and if so, were policy considerations involved. The results of these deliberations are briefly stated in the remarks column of the tabulation.

Although it is not summarized in the tabulation, the outcome of the foregoing procedure was that 22 of the questions were deemed valid policy issues, two were judged to be issues, but more in the purview of other committees, and several were identified as technical assessment problems or invalid issues.

## POLICY STATEMENTS

Of the 22 valid policy questions, the committee had time to develop a position on seven. In the following pages, each of these seven and its accompanying policy statement are briefly stated.

ISSUE: Is there a compliance problem?

REPORT: Monitoring data available from the first nationwide monitoring of public water supplies shows no compliance problems with man-made radionuclides. Substantially all cities impacted by nuclear facilities are in compliance with the Maximum Contaminant Level (MCL) for man-made radioactivity.

Current monitoring data for Ra-226 indicate that about 150 supplies exceed the MCL of 5 pCi/l. However, it has also been projected that about 500 of the 59,812 total water supplies may exceed this MCL which constitutes a compliance problem.

For Ra-228 a possible compliance probelm exists because of undetected violations. Ra-228 concentrations are not determined unless Ra-226 concentrations exceed 3pCi/l. A limited study indicated that 40-50% of Ra-228 violations could go undetected because of this coupling of Ra-228 with Ra-226.

ISSUE: Should EPA adopt a total dose equivalent for the purpose of regulating radionuclides in the drinking water?

REPORT: The Committee regards this as an attractive alternative to setting of MCLs for individual radionuclides and recommends its adoption by the Agency. The total dose equivalent selected should be based on the risk to health but should take into consideration costs and technical feasibility.

Secondary limits in the form of radionuclide concentrations yielding this dose equivalent should be provided by EPA.

ISSUE: Should ICRP-30 calculations and assumptions be used as a basis for revised regulations?

REPORT: The Office of Drinking Water should

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avail itself of the pertinent evaluations and recommendations of recognized radiation protection authorities, such as the National Academy of Science, National Council on Radiation Protection and Measurements, and International Commission on Radiological Protection, for appropriate guidance. An example is ICRP-30 which describes state-of-the-art metabolic and dosimetric models for application to certain radiation protection problems.

ISSUE: Should drinking water standards for radioactivity consider the broad issue of performance of waste disposal sites?

REPORT: The Committee recognized that drinking water standards become a reference point for waste disposal regulations, but its considered view is that for policy reasons laid out in the statute, it is inappropriate to derive drinking water standards to control waste disposal. Equally significant to the Committee is the corollary to this: that EPA in its standards for RCA sites and radioactive waste sites make it clear that the protection needs and authorities for those problems should derive from those problem areas. EPA should not inappropriately apply the drinking water standards to solve waste disposal problems rather than develop requirements appropriate to those problems.

ISSUE: Should gross alpha and beta be used as an MCL or a screening method?

REPORT: The Committee believes that the use of gross measurements as legally enforceable MCLs has inherent problems because those measurements do not reflect appropriate accuracy. On the other hand, they are useful as a screening tool. EPA should provide a framework of MCLs for major specific radionuclides and discontinue the use of gross MCLs. The gross MCLs should be replaced with nuclide-specific MCLs for which monitoring and regulatory actions may be taken on the basis of gross measurements used as a screening mechanism.

ISSUE: Can a method other than monitoring radionuclide concentrations be used to obtain compliance with MCLs such as inspection and certification of components, maintenance records, or demonstration that the source of the drinking water is of high quality?

REPORT: The Committee discussed this issue at some length, expressing that whereas it may be desirable to allow flexibility in achieving compliance, certain practical problems such as system fluctuation and the certainty and public relations worth of measured values may not make it reasonable. Considerable concern was expressed about the technical validity of relying on surrogates such as softness, pH turbidity, etc., as indicators of systems performance and also whether one might not perform more measurements to demonstrate system efficiency than would be required by water-tap-measurements. Having no specific technical factors to allay these concerns, the Committee reached no consensus as to whether this procedure may be achievable despite its seeming desirability. This issue was left unresolved; EPA should examine it further with regard to compliance programs, especially if the frequency of compliance measurements and the number of nuclide-specific MCLs increases.

ISSUE: Two waste-handling aspects exist regarding removal of radioactivity in treatment of drinking water: (1) Does radiation exposure of operators from treatment systems argue against removal of such material and (2) disposal of waste products?

REPORT: For operators, OSHA regulates workplaces, and have to assure that proper procedures are followed, including keeping exposure records. Fundamentally, this is the same issue faced by all standards related to processes that may require filtration or on-line treatment. Control levels are struck on the basis that public health protection is essential even though occupational protection procedures may need to be augmented.

Similarly waste disposal is an accepted necessity associated with all standards requiring on-stream treatment. The Committee believes management of a concentrated waste better than dispersion of material into the environment. For naturally occurring radioactive waste, EPA's RCRA program would need to assure that such collected materials are disposed of properly.

## SUMMARY

The Compliance and Policy Committee feels that the seven issues it addressed are of significant importance in assuring that the health of the public is adequately and equitably protected from insult by radionuclides in drinking water. Further, the brief statements presented herein do not reflect the extent of discussion that led to these statements nor do they reflect the many ramifications which would be involved in implementation of these policies.

The Committee also wishes to emphasize that the fact that policy statements were not developed for the other 15 policy issues considered does not imply that they are necessarily less important. This omission resulted because of limited time available and we recommend that EPA address these issues by subsequent workshops or alternate mechanisms.

## APPENDIX

## TENTATIVE ISSUES CONSIDERED

| | ISSUE | REMARKS |
|---|---|---|
| 1. | Is there a compliance problem with existing regulations? | Valid Policy Issue |
| 2. | If there is a compliance problem, is it measurable in terms of risk, extent, distribution, and costs to correct? | Technical Assessment |
| 3. | Are treatment methods available for non-compliance instances? | Technical Assessment |
| 4. | Does EPA have flexibility within the law to regulate or not regulate? | Valid Policy Issue |
| 5. | Does EPA have a responsibility regarding public education? | Valid Policy Issue |
| 6. | Do small systems require special treatment? | Valid Policy Issue |
| 7. | Do private single family well supplies require special treatment? | Valid Policy Issue |
| 8. | What criteria should be used in making a determination to regulate? | Valid Policy Issue |
| 9. | If EPA has the flexibility to do both, when should health advisories versus MCLs be used? | Valid Policy Issue |
| 10. | Should EPA use a linear or non-linear dose response curve in assessing risk for purposes of establishing standards? | Valid issue but more in purview of other committees. |
| 11. | Is there a threshold for the dose response curve? | Technical Assessment |
| 12. | Should 2 liters/day or some other value be used for water consumption? | Technical Assessment |
| 13. | Should there be protective action guides for water exclusive of other media? If so, how should they be set? | Valid Policy Issue |
| 14. | Are current monitoring frequencies acceptable? | Valid Issue but more in purview of other committees. |

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|---|---|---|
| 15. | Should gross alpha be a screening method or an MCL? | Valid Policy Issue |
| 16. | Can a method other than monitoring radionuclide concentrations be used to ascertain compliance with MCLs? For example, is there a surrogate such as maintenance of treatment plant efficiency? | Valid Policy Issue |
| 17. | Should ICRP-30 calculations and assumptions be used as a basis for revised regulations? | Valid Policy Issue |
| 18. | Should recommended limits be protected of groundwaters at waste disposal sites? | Valid Policy Issue |
| 19. | What is the proper framework for deciding a justifiable level of risk for a given radionuclide in drinking water? | Valid Policy Issue |
| 20. | Should chemical toxicity or radio toxicity be the determining criterion for assessing uranium exposure risk? | Technical Assessment |
| 21. | Should regulations be cast in the framework of a total dose equivalent for members of the public rather than incremental exposures (MCLs) for individual radionuclides? | Valid Policy Issue |
| 22. | Is there a reasonable rationale for treating man-made and naturally-occurring nuclides differently? | Valid Policy Issue |
| 23. | Should EPA use a conservative, middle-of the -road, or liberal philosophy in establishing risk criteria on which to base regulations? | Valid Policy Issue |
| 24. | Is individual, point-of-use, treatment a viable alternative to system treatment for situations of non-compliance? | Valid Policy Issue |
| 25. | Should population risk or individual risk be the determining criterion in standard setting? | Valid Policy Issue |
| 26. | Should internal accumulation models use a time dependent or equilibrium dose rate? | Technical Assessment |
| 27. | Is there a "propagation of conservatism" in the rationale behind current standards? | Technical Assessment |
| 28. | Should only human subject validated models be used for intake and accumulation? | Not a valid issue |

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|---|---|---|
| 29. | Should standards for different nuclides be based on equivalent risks or on varying criteria depending on economics and other factors? | Valid Policy Issue |
| 30. | Should non-community supplies be regulated for radionuclides | Valid Policy Issue |
| 31. | How can predictive modelling which would estimate that radionuclides would not be expected in geographic portions of the U.S. be used in implementation and design of regulations? | Valid Policy Issue |
| 32. | Does the concept of de minimus risk have application in design of drinking water regulations as developed under the Safe Drinking Water Act? | Valid Policy Issue |

## COMMITTEE ON SAMPLING AND ANALYTICAL METHODS

Chairman: Bernd Kahn

Recorder: Seymour Gold

Committee Members: Rolf M.A. Hahne
David McCurdy
William S. Moore
Jacob Sedlet
Stanley Waligora, Jr.
Earl Wittaker

# Radiological Sampling and Analytical Methods for National Primary Drinking Water Regulations

Richard L. Blanchard
Eastern Environmental Radiation Facility
U.S. Environmental Protection Agency
Montgomery, Alabamaa 36193

Rolf M.A. Hahne*
University Hygienic Laboratory
University of Iowa
Iowa City, Iowa 52242

Bernd Kahn
School of Nuclear Engineering and Health Physics
Georgia Institute of Technology
Atlanta, Georgia 30332

David McCurdy and Russell A. Mellor
Yankee Atomic Environmental Laboratory
Yankee Atomic Electric Company
Framingham, Massachusetts 01701

Willard S. Moore
Department of Geology
University of South Carolina
Columbia, South Carolina 29208

Jacob Sedlet
Occupational Health and Safety
Argonne National Laboratory
Argonne, Illinois 60439

Earl Whittaker
Environmental Monitoring Systems Laboratory
U.S. Environmental Protection Agency
Las Vegas, Nevada 89114

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* Currently with Dow Chemical Co., Midland, Michigan

## Abstract

Radiological sampling and analysis performed under the National Interim Primary Drinking Water Regulations were evaluated for the EPA Office of Drinking Water to consider whether any changes should be recommended. The authors reviewed the analytical screening scheme; sample collection, storage, and analysis procedures; selection of analytical methods; reliability of results; and possible future needs.

The main problem in the program has been dependence on a screening scheme of gross alpha particle activity measurement and $^{226}$Ra analysis for predicting elevated $^{228}$Ra levels to determine compliance with the maximum contaminant level (MCL) for radium. In some aquifers, $^{228}$Ra levels have been found to be unrelated to $^{226}$Ra levels. Several alternatives are discussed to eliminate this problem. A secondary problem is that the measurement for assuring compliance with the MCL for gross alpha particle activity minus radium, radon, and uranium utilizes chemical uranium analysis and assumes equilibrium of $^{238}$U and $^{234}$U. Because some ground waters are known to be at disequilibrium, radiometric uranium analysis is needed for those gross alpha particle activities and chemical uranium values that could result in an erroneous conclusion relative to the MCL. In addition, studies were recommended for determining analytical uncertainties and assuring reliable sampling and sample maintenance; improvements in the system for accepting methods were suggested; and methods were identified for several radionuclides not currently in the analytical program that may be needed to assure absence of elevated radiation doses and could be useful for identifying trace contaminants.

## Introduction

The National Interim Primary Drinking Water Regulations (NIPDWR, 40 CFR Part 141), which require determination of specified radionuclides in public water supplies, have been in effect since 1977. Analytical results for tens of thousands of supplies are now available. In 1982, the Office of Drinking Water, U.S. EPA, established committees to advise it concerning the effectiveness of the current Regulations and any changes that might be needed. Reviewed here is the sampling and analysis program.

The regulations can be met by collecting water samples at specified intervals and analyzing either individual samples or composites from each supply according to two analytical schemes. One of these, applied to all samples, determines gross alpha particle activity, $^{226}$Ra, and $^{228}$Ra in that order if the preceding result exceeds the specified screening level. Elevated gross alpha particle activity levels not attributable to $^{226}$Ra may also be analyzed for uranium. The other scheme is for relatively few surface water supplies in large cities and communities downstream from nuclear facilities to determine gross beta particle activity as well as $^{3}$H and $^{90}$Sr; then, if screening levels are exceeded, certain other man-made radionuclides must be analyzed. The schemes are described in detail by another paper in this series (La84).

The results reported so far indicate the extent of the analytical effort required for the continuing program. In 170 of the approximately 47,000 public water supplies for which results have been reported, $^{226}$Ra concentrations exceeded the Maximum Contaminant Level (MCL) of 5 pCi/l; about 350 additional elevated values are expected, although only 13,000 supplies remained unreported (Co83a). Very few supplies exceeded the 15 pCi/l MCL for alpha particle activity less radon and uranium. Occasionally, $^{232}$Th, $^{210}$Po or $^{224}$Ra contributed to the alpha particle activity. Data in a few reports suggest that approximately 250 water supplies may contain $^{228}$Ra in excess of 2 pCi/l (Co83a, Gi81, Ki82, Kr82, Mi80). Sufficient results have been accumulated to indicate that concentrations of $^{228}$Ra are not related to $^{226}$Ra in some aquifers (Gi81, Ki82, Kr82, Mi80). Several investigators have reported that radium concentrations in specific wells remain fairly constant (Gi81, Ho81, Ki82, Kr82, Mi80), with variations only as high as 20%.

In 60,000 drinking water supplies, uranium is projected to occur at the following concentrations (Co83): 25 to 650 exceed 20 pCi/l, 100 to 2,000 exceed 10 pCi/l and 2,500 to 5,000 exceed 5 pCi/l. Most water supplies in which uranium concentrations exceed 5 pCi/l serve small communities.

No man-made radionuclide has so far exceeded its MCL (Co83b). Only a single water supply with a man-made radionuclide at detectable levels -- tritium at a concentration of 3,000 pCi/l (Cl83) -- has been reported.

Elevated concentrations of $^{222}$Rn have been found in a number of underground water supplies (Ki82, He84) in a study unrelated to these Regulations. Some concentrations greatly exceeded levels supported by dissolved $^{226}$Ra. A few measured values of $^{210}$Pb and $^{210}$Po were generally below 1 pCi/l (Ho83). $^{224}$Ra has been looked for and found unsupported by $^{228}$Th (Mo83). Elevated thorium levels have not been found to any extent despite the abundance of thorium in the earth's crust.

The results to date indicate that $^{226}$Ra, $^{228}$Ra, $^{224}$Ra, $^{222}$Rn, and uranium are the only natural radionuclides likely to be in public water supplies at concentrations that may be of health concern. Elevated concentrations of natural radionuclides occur mostly in underground water supplies associated with certain geological formations.

The many results of these analyses (Co83a) reported by individual states, together with published descriptions of and comments on these activities, (Kr82, Mi80, Cl83) permit evaluation of the applicability of these schemes. Results and observations were also reported at the National Workshop on Radioactivity in Drinking Water sponsored by EPA on May 24 - 26, 1983, in Easton, Maryland. Based on this information, consideration was given to the reliability of the currently prescribed sampling and analysis program, its effectiveness in using the best techniques available for the purpose, and the extent to which it provides complete radiological information. Where problems are believed to exist, recommendations are given for changes, additions, or developmental studies.

## Discussion

### Screening Procedures

The currently prescribed regulations for radionuclides in drinking water are found in 40CFR Parts 141.26(a)(1)(i) and (ii) and 141.26(b)(1)(i) and (4)(i) (La84). These separately address naturally-occurring radioactivity and man-made radionuclides. Several problems concerning the screening procedure for the former are discussed here. In the general absence of detectable levels of man-made radionuclides, no recommendations are presented concerning this screening scheme.

The problems concerning the gross alpha particle activity screening scheme are, in order of importance; (1) the scheme does not necessarily indicate whether $^{228}$Ra is at concentrations that contribute to exceeding the MCL for radium; (2) the gross alpha particle activity and $^{226}$Ra concentrations may differ by more than the random error due to counting statistics when only $^{226}$Ra is present; and (3) the gross alpha particle activity and uranium concentrations may differ by more than the random error due to counting statistics when only uranium is present. Several approaches to replacing the screening scheme for $^{228}$Ra are discussed briefly in this section; some procedures that may be sufficiently cost-effective for widespread use as replacements are described in the Analytical Procedures section. Possible improvements for gross alpha particle activity measurement are also discussed, although it should be recognized that this measurement is inherently less reliable than indicated by the counting error. For comparing uranium concentrations with gross alpha particle activity, possible nonequilibrium between $^{238}$U and $^{234}$U is an additional problem.

Radium-228. In the screening procedure, a value of the gross alpha particle activity not in excess of 5 pCi/l is taken to imply a radium concentration not in excess of 5 pCi/l, and a $^{226}$Ra concentration not in excess of 3 pCi/l implies a $^{228}$Ra concentration not in excess of 2 pCi/l. In the absence of other information, the $^{226}$Ra:$^{228}$Ra ratio was expected to be 3:2 or greater, so that water systems with $^{226}$Ra not exceeding 3 pCi/l would not exceed the 5 pCi/l MCL for these two radium isotopes of interest. This has proven not to be the case.

Ratios of $^{226}$Ra:$^{228}$Ra ranging from over 14 to less than 0.07 have been reported (Mi80, Kr82, Ki82) and values below 3:2 are relatively common. Thus, the screening scheme can not be depended on to assure the absence of total radium concentrations above 5 pCi/l unless the $^{226}$Ra:$^{228}$Ra ratio can be predicted for a particular aquifer.

The screening scheme was developed to avoid requiring $^{228}$Ra analyses for numerous samples. The $^{228}$Ra method (Kr76) specified in Part 141.25 is relatively long and expensive. Neither $^{228}$Ra nor its short-lived daughter, $^{228}$Ac, emit alpha particles for detection by the gross alpha particle activity measurement. The next radionuclide in the decay chain, $^{228}$Th, emits alpha particles but accumulates slowly because of its 1.9-year half life. It may also be deposited on container walls because of the insolubility of thorium. Its daughter, $^{224}$Ra, emits alpha particles but is generally associated with $^{228}$Th because of its relatively short half life (3.64 d).

In the absence of any nationwide $^{226}$Ra:$^{228}$Ra ratio, one of the following three approaches should be considered for replacing the 3:2 ratio assumed in the screening scheme:

(1) A national study of aquifers that contain $^{228}$Rn could permit implementation of the requirement that $^{228}$Ra be measured where its presence is expected. Even a few regional studies might be sufficient to demonstrate patterns of elevated $^{228}$Ra levels for predicting aquifers with such elevated levels.

(2) Review of available $^{228}$Ra or total radium procedures (see Analytical Procedures) may permit selection of a method that has acceptable levels of effort and cost for processing every sample.

(3) One or more screening procedures for $^{228}$Ra or total radium may be found for replacing the current screening scheme. For example, gross beta particle activity measurement or total radium measurement after selected intervals for ingrowth of

$^{226}$Ra and $^{228}$Ra progeny could be applied if their detection limits were sufficiently low.

Radium-226. The results of the gross alpha particle activity measurements may differ from $^{226}$Ra results in the absence of other radionuclides by more than the specified 95% confidence level value based on random counting error because several sources of error are currently not considered. These errors can occur because (1) the detector is calibrated with a different radionuclide, (2) the correction for self-absorption is only approximate, or (3) short-lived progeny of $^{226}$Ra accumulate to various degrees in the sample for gross alpha particle activity. Hence, the requirement in Section 141.26(1)(ii) that $^{226}$Ra be measured if the gross alpha particle activity exceeds 5 pCi/l may result in overlooking some water supplies in which the $^{226}$Ra concentration exceeds the MCL by several pCi/l. Suggestions for improved measurements when the total solid content of a sample is high and for using specific standard radionuclides are given below. Beyond this, the gross alpha particle activity must be recognized as an estimate.

Uranium. For $^{238}$U plus $^{234}$U radioactivity determined by chemical uranium analysis and calculated by multiplying the analytical result in micrograms by the equilibrium factor of 0.67 pCi/μg, the result will be in error if the two isotopes are not in equilibrium. The actual factor can range from 0.33 pCi/μg (no $^{234}$U) to at least 7 pCi/μg ($^{234}$U:$^{238}$U = 20). Hence, the calculated gross alpha particle activity, excluding $^{226}$Ra, $^{222}$Rn and uranium, could be in error if based on chemical determination of uranium and assumption of equal activity of $^{238}$U and $^{234}$U. At $^{234}$U:$^{238}$U ratios below 1.0, the uranium activity is overestimated and the resulting "gross alpha particle activity minus uranium" is falsely low. The above comments concerning the reliability of the gross alpha particle activity also apply here.

### Analytical Procedures

Currently approved analytical methods for drinking water compliance monitoring are referenced in the NIPDWR and are published in EPA's Interim Radiochemical Methods Manual (Kr76), Standard Methods (APHA71, ASTM75), or the HASL-300 Manual (Ha75). The following improvements are suggested for consideration.

Gross alpha and gross beta particle activity. The national program has relied heavily on gross alpha and gross beta particle activity screening of water samples. The current practice of distributing two unspecified radionuclides combined for gross alpha and beta particle activity in the EPA cross-check program for radionuclides in water, however, can result in consistent calibration error. Performance evaluation studies (De83) show that numerous laboratories have had problems with gross beta activity measurements of cross-check samples that contain the usual $^{241}$Am and $^{137}$Cs. Reliability of results might be improved if the radionuclides in these cross-check samples were selected for calibrating detectors, and were identified so that the user could determine their suitability for a specific isotope calibration.

A pure alpha-particle emitter such as $^{230}$Th is recommended as a standard and cross-check solution for gross alpha particle activity. The $^{241}$Am currently used in these cross-check samples emits numerous conversion electrons and soft X rays that are detected as beta particles and can be attributed to the beta particle emitter. The $^{137}$Cs used in the same solution for gross beta particle activity is acceptable if the

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radionuclide is identified so that the presence of an additional 9.5 percent conversion electrons from $^{137m}$Ba can be considered in calibrating for other isotopes.

Some public drinking water supplies have relatively high dissolved solids levels, which cause self-absorption of alpha particles and greatly reduce the sensitivity of the gross alpha particle activity measurement. For a gross alpha activity screening test with a 20-cm$^2$ planchet, a dissolved solids concentration limit of 500 mg/l (25 mg/cm$^2$) is recommended. Barium sulfate/iron hydroxide coprecipitation (Li82) may be considered for gross activity measurement if samples with higher dissolved solids are involved. This method is being evaluated by EMSL-Las Vegas. In the range 0 - 25 mg/cm$^2$, EPA could assist in improving data reliability either by determining self-absorption factors for the more common alpha-particle activity detectors or by distributing calibrated solutions for performing this determination.

Radium-228. At present, two approved test procedures can be used nationwide for measuring $^{228}$Ra concentrations in drinking water samples. Both methods are technique-dependent, time-intensive and costly. Many laboratories that participate in the EPA EMSL-Las Vegas intercomparison and performance studies for $^{228}$Ra have been using the method referenced in the NIPDWR (Kr76). Tables 1 and 2 show the high fraction of unacceptable $^{228}$Ra results during four-year periods of intercomparison and performance tests.

Another problem with the $^{228}$Ra method is possible contamination by the $^{90}$Y daughter of $^{90}$Sr. Tables 1 and 2 show the interference when the samples are analyzed by the approved method. Performance samples in Table 1 contain a complex mixture of radionuclides, including $^{90}$Sr-$^{90}$Y.

Intercomparison samples in Table 2 contain only $^{226}$Ra and $^{228}$Ra plus their progeny, except when 25 pCi of $^{90}$Sr per liter were added in the 9/79 study to examine the effects of $^{90}$Y daughter interference.

Other methods that are becoming available appear to be improvements over the approved methods (No81) and should be evaluated. Many of them simultaneously determine $^{226}$Ra. One such method uses beta-gamma coincidence counting after radium coprecipitation with barium sulfate (Mc81a). The method requires a 1-liter sample and minimal chemistry in order to meet the required sensitivity of measurement for $^{228}$Ra. The instrument consists of a well-type NaI(Tl) detector for gamma rays, a thin plastic scintillator for detecting beta particles, and a coincidence pulse analyzer.

Another method also coprecipitates radium with barium sulfate, then dissolves the precipitate in alkaline EDTA, mixes the solution with liquid scintillator solution, and counts the low-energy beta particles of $^{228}$Ra (As81). The author claims adequate sensitivity with one liter samples. A modification of this method has been tested by EPA EMSL-CIN (Ve83).

A third method uses a 3-liter sample and the same radium coprecipitation. It collects the precipitate on a filter, allows two days for $^{228}$Ac ingrowth, and then analyzes for $^{228}$Ra with a gamma-ray spectrometer. The 986-keV gamma-ray emitted by the ingrown $^{228}$Ac is measured. The State of Arkansas is seeking approval for use of this method (He82).

In a fourth method, radium is collected from a multiliter sample on a column of fibers coated with manganese dioxide (Mi81). Radium is then dissolved from the column, coprecipitated with barium sulfate, collected on a filter paper, and analyzed for $^{228}$Ra by counting gamma rays emitted by the ingrown $^{228}$Ac, as in the above-cited method. After a period of weeks to months, the ingrown $^{228}$Th plus $^{224}$Ra can also be measured with an alpha-particle detector. As an alternative, the fibers are compressed and $^{228}$Ac is counted directly with a gamma-ray spectrometer.

Uranium. According to Section 141.15 part (b) of the NIPDR, if the gross alpha particle activity exceeds the $^{226}$Ra concentration by 15 pCi/l, then a uranium determination is necessary. The uranium measurement referenced in the NIPDWR is a fluorometric method which measures uranium mass (ASTM75).

Because $^{234}$U:$^{238}$U isotopic ratios have been found to vary in groundwater systems from less than 1 to 20 (Os82), the factor of 0.67 pCi/$\mu$g for a 1:1 ratio may not apply. Therefore, the fluorometric method can only be relied upon to give the concentration of uranium in $\mu$g/L. If the radioactivity level is needed, the revised regulations should refer to a radiochemical method that measures the total uranium alpha particle activity of the sample. Such a method has been published (Kr80). The method has been multilaboratory tested in a collaborative study and precision and accuracy have been estimated.

If the gross alpha particle activity in excess of the measured $^{226}$Ra concentration is consistent with the uranium fluorometric (mass) measurement at a 0.67 pCi/$\mu$g conversion factor, then results of the fluorometric method are acceptable. Moreover, some uranium measurements in ground water suggest that ratios greater than 5:1 usually occur at low concentrations (1 pCi/l and less) (Os82). Under these circumstances, the ASTM method or several alternatives are usable, although any such alternatives would need official method approval. The Prescribed Procedures (Kr80) have a carbonate/fluoride fusion fluorometric method for uranium with a detection limit of 0.1 μg/l. With laser excitation, the detection limit without purification or concentration is 0.05 μg/l (Pe82).

Iodine-131. The MCL for $^{131}$I in the NIPDWR is 3 pCi/l and the required sensitivity of measurement is 1 pCi/l. Gamma spectrometric analysis of drinking water samples usually is not sufficiently sensitive without concentrating the $^{131}$I in the sample. A radiochemical concentration method for $^{131}$I that has sufficient sensitivity was included in EPA's Interim Radiochemical Methodology (Kr76) and repeated in Prescribed Procedures (Kr80). When that method was multilaboratory tested in a collaborative study, however, low yields were identified as a problem.

Strontium-90. The $^{90}$Sr method (Kr76) still appears to be difficult for many laboratories (EPA81) despite many years of use. Removal of calcium interference with concentrated nitric acid may be the major problem because the acid concentration is crucial in separating strontium from calcium by solubility difference. Other published methods, for example, purification of strontium by ion exchange (Po67), could be evaluated as alternatives.

### Sensitivity and Uncertainty

The NIPDWR (EPA76) addresses these topics only briefly, indicating in Section 141.25 (c) that the listed detection limit is the concentration which can be counted with a precision of ± 100% at the 95% confidence limit (1.96 times the standard deviation of the net counting rate of the sample.) Section 141.25 (c) also specifies that the detection limit for the combined $^{226}$Ra and $^{228}$Ra concentration (MCL is 5 pCi/l) shall not exceed 1 pCi/l and for gross alpha activity (MCL is 15 pCi/l) the detection limit shall not exceed 3 pCi/l.

Additional information on sensitivity, accuracy, and uncertainty is given in the EPA's Prescribed Procedures (Kr80). For each procedure (except actinides), the calculated detection limit and the precision and bias determined in a collaborative or a single-laboratory replicate test study are given. The equation for calculating the counting error and three methods for determining the counting time required to obtain the required sensitivity are given in appendices to that report.

Under the current drinking water regulation, uncertainty is not a factor in determining compliance, except in one instance. According to Section 141.26(a)(1)(i), gross alpha particle activity may be substituted for radium analysis if the result does not exceed 5 pCi/l at a confidence level of 95 percent. Otherwise, Section 141.25 (d) states that "to judge compliance with the maximum contaminant levels ... averages of data shall be used and shall be rounded to the same number of significant figures as the maximum contaminant levels."

To characterize the reliability of results, detection limits and uncertainty of methods applied under normal conditions should be determined and reported by each participating laboratory. The methods of calculating uncertainty (Be69, Cu78, Ma78) and the detection limit need to be described by the EPA in greater detail than given in Section 141.25(c) to assure consistent application by individual laboratories. It is important to include not only the random counting error as is now done, but also random and systematic errors in sample collection and storage, chemical procedures, and detector calibration and use.

Analytical uncertainty can be determined from the laboratory cross-check program, method validation studies, and tests with known sources, blanks, and replicate samples within a laboratory. When sufficient empirical data have been obtained, the EPA should replace its currently specified detection limits based on counting precision with limits based on these measurements. It should also use this information to determine whether analytical laboratories report results with acceptable analytical reliability, and whether the results for cross-check samples in the quality assurance program are good indicators of reliability.

### Acceptance Criteria for Alternative Methods

Within Section 141.25, referenced analytical methods are stipulated which must be employed in order to comply with regulations. Section 141.27 states that an alternative method is acceptable "if it is substantially equivalent to the prescribed test in both precision and accuracy as it relates to the determination of compliance with any maximum contaminant level." Other than the exceptions noted in the regulation, no other radiochemical procedure is considered acceptable without obtaining the written permission of the Administrator of the U.S. EPA and the appropriate agency of the State where the laboratory is located. Initially, the EPA only considered approving alternative techniques according to a very strict protocol, i.e., NIPDWR nationwide approval so that all laboratories throughout the country could benefit and utilize the technique.

Within the past few years, granting alternative method approval has been modified to embrace "limited" approvals. For testing a "limited alternative technique", the total number of analyses is 120 (60 analyses by the alternative technique and 60 analyses by the approved methodology) as compared to 240 analyses on a national basis. This "limited" approval permits that laboratory to utilize the alternate methodology for water samples from the same watersheds as the test water. In both types of approvals, the data are evaluated for equivalency according to standardized statistical methods (EPA77). This, however, evaluates the accuracy of the alternative technique only if the originally approved technique is adequate.

Only one radiochemical method has been given an NIPDWR nationwide alternative method approval status by the EPA since the promulgation of the regulations. Approval for this method, to analyze $^{228}$Ra, was based on its technical equivalency to the approved technique and not on the comparability testing criteria.

Statements concerning measured precision and accuracy parameters are presented for most procedures in the Prescribed Procedures manual (Kr80) intended to update the earlier EPA manual (Kr76), although the former has not been officially accepted as a replacement. These statements indicate that several of the procedures have only been evaluated for accuracy and precision by a single laboratory test involving, in general, nine analyses of a single solution containing a known amount of the radioisotope of interest in a matrix of unspecified chemical composition. Thus, in the case of Methods 900.1, 901.0, and 907.0, the accuracy and precision capabilities of the methods may not be well known or acceptable.

No definite precision and accuracy criteria seem to have been applied to the originally approved methods. Moreover, no relationship appears to exist between precision and accuracy criteria for the EPA Intercomparison Studies Program (EPA81) currently used as a compliance measure, and the measured parameters for the procedures given in the Prescribed Procedures manual.

The EPA has also utilized the protocol for interim certification (EPA78) to test the capability of laboratories and ensure data quality. This program consists of inspecting the radiochemical laboratory every three years and requiring participation in the EPA interlaboratory cross check program. The results of this program, however, have not been used for laboratory certification or decertification.

In brief, the existing system makes it extremely difficult to introduce new methods (EPA77a), although currently approved methods are not as well defined for accuracy, precision, and sensitivity as would be required for a new method. The inspection and cross-check program identifies unsatisfactory conditions but does not necessarily eliminate them. A much more desirable framework for this extensive national program would encourage development and application of improved methods while assuring continued reliability and uniformity of data produced in numerous laboratories. Two alternatives for the current system of methods approval are suggested here, one that substitutes laboratory accreditation and the other that provides a definite test of reliability.

The preferred option is a change to laboratory accreditation. A currently active Federal activity that appears applicable is the National Voluntary Laboratory Accreditation Program (NVLAP)(DC76, DC79) administered by the National Bureau of Standards. This program is being applied in the radiometric measurements field to personnel radiation dosimetry processing and is being evaluated for accrediting in-vivo and in-vitro bioassay processors whose clients are regulated by the Nuclear Regulatory Commission. The concept of the NVLAP for radiation dosimetry

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processing is particularly pertinent since the ability to process personnel radiation dosimeters routinely is being accredited, regardless of the techniques used to measure the radiation dose.

The general NVLAP program encompasses all efforts necessary to ensure that routinely produced data reflect the capability of the laboratory which was demonstrated under performance testing. These efforts include administering the performance testing program according to an established standard or methodology, on-site peer evaluations of laboratories to assess their capabilities, and publishing a list of accredited facilities. Procedures exist for submitting recommendations by a peer review committee, removing accreditation for inadequate performance, performing additional onsite evaluations, and reaccrediting facilities.

As a secondary option, the EPA can modify the existing protocols for alternate method approval to expedite the process for both single-laboratory and nationwide-use approvals. In this approach, each procedure is submitted by the applicant in a detailed format for review.

For single laboratory approval, a set of representative water samples containing known quantities of the radionuclide to be measured are supplied by the EPA Quality Assurance group to the applicant laboratory for replicate analysis. These samples contain the radionuclide (1) near the detection limit, (2) near the MCL, and (3) at a higher concentration to determine accuracy and precision reliably. Also included in this set are (4) a blank sample and (5) one or more samples that contain the radionuclide plus potentially interfering stable and radioactive materials. The result of these analyses must satisfy published criteria of acceptance for accuracy, precision, and sensitivity.

For approval nationwide under this option, a second laboratory must qualify for approval in the use of the same method. Subsequently, a multi-laboratory collaborative study must be conducted, with the results again meeting predetermined levels of acceptability.

### Sampling Frequency

The monitoring frequency for naturally-occurring radionuclides is specified in 40CFR Part 141.26(a). Although there is evidence that radium in some ground-water sources remains at relatively constant levels (Ho81, Ki82, Kr82, Mo83), significant changes in the radiochemical composition of the water could occur for a given well due to changes in pumping rates or ground-water levels. Examination of data from repeated samplings is recommended to determine whether the currently specified 4-year cycle of reanalysis is appropriate.

### Sample Stability

The NIPDWR allow quarterly sampling and analysis, averaging for the annual concentration, or compositing quarterly samples and an analysis of the composite for the annual average concentration. The integrity (stability) of such samples stored before analysis needs to be evaluated for the radionuclides specified in the Regulations. Physical changes such as precipitation have been observed by EPA (EMSL-Las Vegas) personnel during on-site laboratory evaluations for certification in some stored drinking water samples, even when samples were acidified to pH 2 or lower.

Sample preservation procedures have been recommended in methods manuals that are referenced in the NIPDWR and the EPA certification manual (EPA78). The preface to the EPA methods manual (Kr76) suggests that "when quarterly composites are set aside for future analyses, the samples should be acidified with 1 ml 16 N $HNO_3$ per liter to minimize losses caused by adsorption on container walls." Chapter 6 of the EPA certification manual recommends that drinking water samples be acidified to pH 2 at the time of collection. If this cannot be done in the field because samples are to be separated into soluble and insoluble fractions, then samples must be acidified upon arrival at the laboratory followed by a holding period of at least 16 hours (overnight) before the samples are analyzed.

Section 105 of Standard Methods (APHA80) and several EPA publications (EPA79, Be82) recommend preservation procedures while indicating that complete sample stability by preservation treatment is practically impossible. Some preservation treatments are effective for specific substances, but in many cases they only retard chemical and biological processes that continue after the sample is collected.

Some information on the effects of long-term sample storage is available. A survey of the literature on long-term storage of environmental samples to be analyzed for trace elements (Ma76) suggests that information on sampling and storing trace elements in environmental samples should also apply to radionuclides. One study (Ei65) used dilute aqueous solutions of radionuclides to determine the relative adsorption of radionuclides on glass and plastic surfaces. A study of reactor water and waste solutions collected for analyses demonstrated the fractionation of radionuclide ions among the soluble and insoluble portions and the walls of the container (Be80).

Although the studies contain some conflicting details, they show that loss of trace elements, including radionuclides, to container walls can be a serious problem for water samples. Losses have been reduced with reagents that tend to maintain a substance in its soluble form, such as strong acids or complexing agents; with isotopic carriers to prevent "radiocolloidal" behavior; and with substances that tend to saturate ("pacify") adsorption surfaces. Containers with a less adsorptive surface or surface coatings that reduce adsorption have also been found. Such remedial measures, however, usually apply to a specific radionuclide rather than the entire group of radionuclides to be analyzed. Worse, a good preservation technique for one radionuclide may cause losses for another.

## Sampling Reliability

In view of the time and cost of sample analyses, commensurate efforts should be applied to assuring that the sample adequately represents the water supply. The water must be collected at uncontaminated taps in uncontaminated containers, from a freely flowing supply. The sample must represent the water used throughout the distribution system, from the usual sources. Conditions of reproducibility and absence of contamination can be encouraged by preparing collection protocols.

Sampling each source of water in a multi-source system, although an additional effort, would provide definition of all possible combinations of sources. At present, Section 141.26(a)(3)(iii) requires analysis of all sources only if ordered by the State. Quantification of the radionuclide content in the various sources would permit calculation of concentrations after the application of measures such as mixing, special treatment, or termination of some sources to obtain acceptable levels.

Possible effects of the distribution system on radionuclide levels at the tap should be evaluated for systems in which radionuclide levels are elevated at the source. Differences in radionuclide levels throughout a system can be caused by connections to sources at various points; removal of radionuclides by deposition, volatilization and radioactive decay; or remobilization of radionuclides accumulated at some points in the system.

## Expanded Analytical Program

If the EPA considers potential radiation doses from elevated levels of uranium and radon, it may specify MCL values for these radionuclides and require analyses with prescribed methods. Other reports in this series discuss the radiation doses associated with uranium and $^{222}$Rn in water. The dose from $^{234}$U plus $^{238}$U appears to be of the same order of magnitude as from $^{226}$Ra at the same concentration. Although the dose per pCi of $^{222}$Rn is much lower, the concentration of $^{222}$Rn in some ground waters is much higher than $^{226}$Ra. Persons may be exposed to radiation from $^{222}$Rn in the water supply by drinking the water or inhaling $^{222}$Rn that has escaped from water into room air. In either case, the important source of radiation exposure is alpha particles emitted by short-lived $^{218}$Po and $^{214}$Po progeny of $^{222}$Rn.

The volatility and short half life of $^{222}$Rn differentiate this radionuclide from the natural radionuclides now measured under the Regulations. These characteristics will result in lower $^{222}$Rn concentrations at the tap than measured at the source; permit application of relatively simple removal techniques; and introduce sources of error in analysis.

Methods for analyzing the naturally occurring radionuclides of thorium and polonium may be needed on a few occasions to examine water with an elevated gross alpha particle activity not attributable to radium, uranium, or radon. Because of their relative insolubility in water under most environmental conditions, these radionuclides ($^{234}$Th, $^{232}$Th, $^{231}$Th, $^{230}$Th, $^{228}$Th, $^{227}$Th, and $^{210}$Po) would normally not be encountered at concentrations that exceed the alpha particle screening levels. In only a few instances among the tens of thousands of samples analyzed so far were $^{232}$Th and $^{210}$Po detected at levels exceeding the screening value for the gross alpha particle activity.

The following methods can be considered for these analyses:

Uranium. Fluorometric methods for determining uranium by mass and radiometric methods have been discussed in a preceding section. Although no MCL for uranium is currently given, these methods appear to have the necessary sensitivity and accuracy if the MCL were 10 pCi/l or higher.

Radon. Methods for the determination of $^{222}$Rn in water include de-emanation into a scintillation flask or Lucas cell (Lu57), gamma spectrometry (Lu64), high volume extraction followed by liquid scintillation counting (No64, Ho77), and direct low-volume liquid scintillation counting (Pr77, Ho83). The latter method is probably the most rapid and simple, while other methods may exhibit higher sensitivity. Concentrations of $^{222}$Rn at several hundred pCi/l have been measured precisely and accurately with the direct, low-volume, liquid scintillation counting procedure (Ho83). It would be especially suited for large numbers of samples if the MCL were in the hundreds or thousands of pCi/l. This method has been used extensively by EPA (EPA78a).

One possible defect in the present method being used by the EPA is the collection of a 10 ml aliquot by drawing (negative pressure) the aliquot into a syringe and then transfering the aliquot to a liquid scintillation vial which contains the mineral-oil-based scintillator solution. This may draw radon out of solution and cause some loss during transfer to the scintillation vial. Filling the vial to a 10 ml premarked level under positive pressure by transfering the aliquot from a free flowing tap with a needle attachment should be considered.

A standard $^{222}$Rn generating source is being prepared for EPA (EMSL-LV) by the NBS. The above-cited method (EPA78a) will be multilaboratory tested when the standard becomes available.

Thorium. A method for alpha-particle-emitting actinides with individual elements separated sequentially is in the EPA Prescribed Methods manual (Kr80). Separation of thorium by coprecipitation and solvent extraction is part of this procedure and can be used separately or to determine several actinides simultaneously. Testing this procedure to determine reliability and precision is planned by EPA.

Polonium. Most $^{210}$Po procedures, with minor variations, recover polonium from solution by electrochemical displacement onto a metal planchet that is more electropositive than polonium. Silver or nickel are the metals generally used (Ba66, Ba75, Bl66, Ho66, Hu70, Li66). Separation of polonium by other techniques, such as precipitation (Ru66) or ion exchange (Da57), are rarely used. Spontaneous deposition of polonium onto nickel or silver is a reliable technique with recoveries generally exceeding 90 percent. This procedure is also rapid and simple.

In the recommended procedure (Bl66), a 1,000-ml sample of acidified drinking water is evaporated to 100 ml. A nickel planchet is placed in the sample. The polonium deposits on it while the solution is being stirred at 85° C for 3 hours. The alpha particle count rate is measured on the rinsed planchet, and the disintegration rate is computed for an estimated yield.

## Summary and Conclusions

The radionuclide analyses of public water supplies performed under the NIPDWR and parallel state regulations have yielded extensive nationwide information concerning the absence of elevated levels in most systems and the occurrence of elevated radium and uranium levels in a few areas. These efforts and some ancillary studies also provide information that may be applied to evaluating the reliability of the data, suggesting improvements in procedures, planning studies to resolve uncertainties, and possibly expanding the program for better defining the radiation dose.

The gross alpha particle activity screening scheme for naturally occurring radionuclides in water has been very useful. It may miss elevated $^{228}Ra$ concentrations, however, lead to an erroneous value of "gross alpha particle activity excluding uranium and $^{222}Rn$", and give a different gross alpha particle activity than $^{226}Ra$ concentration even in the absence of any other radionuclides. The first problem is the most significant one associated with NIPDWR results. A development effort is recommended to replace the screening scheme with a procedure that will assure determination of $^{228}Ra$. Such procedures are available, but factors of time, cost, and applicability by numerous laboratories must be considered in selecting an improved approach. The second problem is due to disequilibrium between $^{238}U$ and $^{234}U$ in some aquifers, whereas the recommended chemical analysis of uranium determines only $^{238}U$ and assumes equal activity of $^{234}U$. Use of any one of several available radiometric methods for the few samples in which this source of error could lead to a value exceeding the MCL would eliminate the problem. The third problem is due to the lesser reliability of the gross alpha particle activity measurement compared to $^{226}Ra$ analysis. Its reliability can be improved by use of defined alpha particle activity standards and assistance in determining self-absorption corrections.

Little can be said concerning gross beta activity screening for man-made radionuclides. No MCL values were exceeded -- in fact, positive results were rare.

A number of approved analytical methods can bear improvement, especially the method for $^{228}Ra$. The existing system for approving new methods is so cumbersome, however, that it discourages improvement efforts. Two alternatives are offered to replace the existing approval system. The preferred replacement is laboratory accreditation, which could be combined with the current EPA laboratory evaluation program through inspection and distribution of cross-check samples. The other alternative requires that laboratories obtain acceptable analytical results for sets of approval samples distributed by the EPA quality assurance program. Either approval program needs to maintain a careful balance between assuring that analytical results from all participating laboratories are reliable and encouraging improvements in methods.

Several aspects of the program were identified for further attention in the future. These include preparing a sample collection protocol to assure reliable sampling; evaluating the impact of source and distribution system variables on radionuclide levels at the tap; examining the stability of the radionuclides of interest in stored water samples to assure minimal losses; and better defining the collected information in terms of standard deviations and detection limits applicable to the reporting laboratory.

Analytical procedures were recommended for $^{222}$Rn, radiometric uranium, thorium, and polonium. The first two radionuclides would require routine analysis if EPA recommends an MCL for each. The latter two have been found only rarely in water supplies, but analyses would be useful if elevated gross activity levels can not be attributed to other naturally occurring radionuclides.

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